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PUBLISHED BY
THE LARYNGOSCOPE
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THE
LARYNGOSCOPE.

VOL. LXIII FEBRUARY, 1953 No. 2

THE CAPILLARIES OF THE VESTIBULAR MEMBRANOUS LABYRINTH IN THE GUINEA PIG.*

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INTRODUCTION.

Our knowledge of vascular ramifications within the vestibular labyrinth has changed little in the past 50 years. The first comprehensive description and illustrations of the main circulatory features of the human vestibular labyrinth were given by Siebenmann[1] in 1894. His studies were so well executed that 10 years later there was little for Shambaugh[2] to add to the vestibular circulation in his observations on the domestic pig. Asai's[3,4] investigations, in 1908, and those of Nabeya,[5] in 1923, confirmed the findings in other mammals and revealed certain species variations.

Capillary relations have been less clearly defined. Siebenmann[1] noted the presence of diffuse, subepithelial networks in the maculae and cristae. Shambaugh[2] added a description of the vessels of the semicircular canals. Nabeya's[5] notes on the capillaries are vague and brief. In general, the capillaries have been dismissed as "complex networks."†

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†After this paper had been prepared for publication, a supplement concerning the vascularization of the human labyrinth by Scuderi and del Bo[6] in the Arch. di Otol., Rinol. e Laringol. was received. Some of their observations on human material are similar to those described in the present studies on the guinea pig's labyrinth.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Dec. 8, 1952.

Previous investigations[7] have shown an orderly arrangement of the capillaries in the cochlea of the guinea pig. It was thought that comparable studies of the vestibular labyrinth might reveal a similar disposition of the finer vessels. Whatever the vascular arrangement might be, the circulation for the cochlear and vestibular labyrinth must be considered together if our knowledge of capillary ramifications within the internal ear is to be complete.

MATERIAL AND METHODS.

Twenty-five guinea pigs weighing from 250 to 400 gm. were used. They were young, healthy animals of dark color. Most of these were studied after intravascular precipitation of Prussian blue or lead chromate by methods previously described.[7] The vessels were examined *in situ* under the stereoscopic microscope at magnifications of 9× to 144×. Pieces of membranous labyrinth were dissected free, counterstained with 0.2 per cent aqueous Safranin and mounted for examination under higher power of magnification. Sections of paraffin and celloidin embedded material were also studied.

FINDINGS.

The vestibular membranous labyrinth in the guinea pig is supplied by two main arteries: the A. vestibuli anterior and the A. vestibuli posterior (see Fig. 1).

I — The A. Vestibuli Anterior.

The origin of the anterior vestibular artery is variable in the guinea pig. It may be either a branch of the labyrinthine artery or of the anterior inferior cerebellar artery. The latter pattern was found more frequently in the animals examined.

The artery follows the bony ridge between the cochlear and the superior vestibular portions of the acoustic nerve, giving off a few small branches to the dura, the vestibular ganglion and occasionally to the facial canal. It enters the bony canal with the fibres of the superior vestibular ramus and pursues a twisted course between bone and nerve, giving off many small and an occasional large branch to the nerve bundle.

Four main branches from the anterior vestibular artery enter the vestibule along the anterior wall. The first large branch usually arises at a point just central to the macula cribrosa for the utricular nerve. This branch is variable and may be absent. It circles the nerve fibres to reach the vesti-

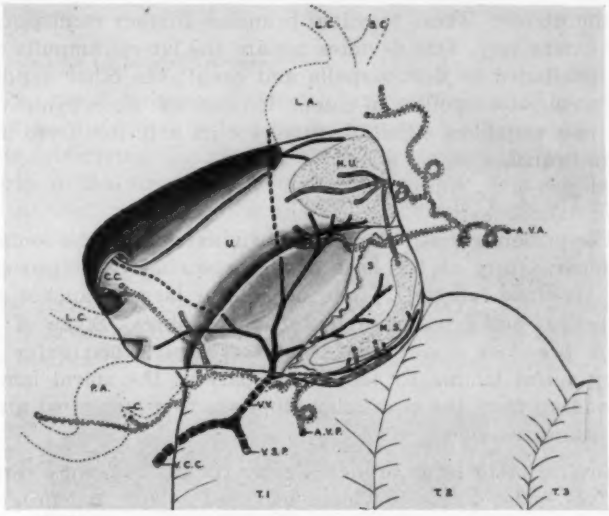

Fig. 1. Photograph of a schematic drawing showing the main arterial and venous features of the circulation of the membranous vestibular labyrinth in the guinea pig. The bone around the oval window and the lowermost part of the basal turn of the cochlea have been removed to expose the vestibule from its inferolateral aspect. (A.V.A.) A. vestibuli anterior; (A.V.P.) A. vestibuli posterior; (C) crista; (C.C.) common crus; (L.A.) lateral ampulla; (L.C.) lateral canal; (M.S.) macula sacculi; (M.U.) macula utriculi; (S.C.) superior canal; (T1) basal turn of the cochlea; (T2) second turn of the cochlea; (T3) third turn of the cochlea; (U) utricle; (V.C.C.) V. canaliculi cochleae; (V.S.P.) V. spiralis posterior; (V. V.) V. vestibularis.

bule and descends in the bone between utricle and saccule. At the edge of the utricle it leaves the bone and is distributed mainly to the under surface of the utricle.

The second branch originates opposite the macula acustica of the utricle. It divides, coiling as it turns around the nerve in the canal, and enters the bone above the macula. Here the branches ramify further before descending toward the utricle.

Most of the arterioles end in the capillary network of the macula; one or two go to the saccule, and a few bypass the neuroepithelial region to enter the network encircling the utricular sac.

The artery continues to coil in the canal with the nerve fibres for the lateral and superior ampullae, dividing superior to the utricle. These terminal branches further ramify, coiling extensively. One deviates toward the lateral ampulla and is distributed to that ampulla and canal; the other supplies the superior ampulla and canal. Because of the proximity of the two ampullae, either may receive an arteriole from both main branches.

II — The A. Vestibuli Posterior.

The posterior vestibular artery originates from the common cochlear artery at the base of the modiolus. It approaches the vestibule, giving off one or several large branches and numerous small branches to the nerve fibres. Some of the large branches divide and twist tortuously, then enter the bony spiral lamina to supply the part of the spiral lamina extending from the vestibular blind sac to a point just above the round window.

Several other large branches enter the inferior bony rim of the vestibule. These coil tortuously in the bone and progress along the anterior border of the saccule. At intervals they straighten out, sometimes divide further and descend toward the neuroepithelium.

The artery then enters the vestibule. It crosses in the bone behind the vestibular vein in its approach to the posterior ampulla. A large vessel branches off to course in the vestibular wall toward the utricle. This usually gives a small branch to the utricle at its anterior edge, while the main artery continues in the bone to the mouth of the common crus. Here a small twig may leave the vestibule with the endolymphatic duct, then the main arterial branch turns into the common crus and is divided between the posterior and superior canals.

The posterior vestibular artery makes its terminal ramifications near the vestibular blind sac. One part coils exten-

sively and divides into arterioles which supply the vestibular scala media. A branch is given to the lateral canal, and the artery then terminates in the posterior ampulla and canal.

All of these large vessels described above usually course through the vestibule in shallow bony channels until they reach the area of the membranous labyrinth they are to supply.

III — The Capillary Areas.

The capillary areas of the membranous vestibular labyrinth may be divided into two main groups: the dense capillary beds underlying the neuroepithelial areas and the loose networks in the utricle and semicircular canals. The capillaries of the vestibular ganglion and the nerve fibres, though not a part of the membranous labyrinth, are also included.

1. The Utricle. The macula acustica of the utricle is supplied entirely by the anterior vestibular artery. The arterioles from its second branch coil in the bone lateral and anterior to the macula (see Fig. 2). The space they occupy contains loose

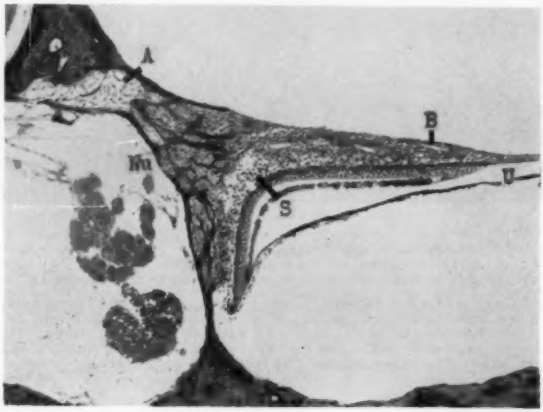

Fig. 2. Photomicrograph of section through the macula of the utricle showing the artery coiled in the bone above the nerve fibres, and the subepithelial capillary plexus of the macula; 15 microns thickness; Heidenhain Susa fixation; Harris hematoxylin and eosin stain; magnification 75×. (A) Arterioles coiled in the bone above macula; (Nu.) N. utricularis entering vestibule; (B) vessel to loose network of utricle; (S) subepithelial capillary plexus; (U) utricle.

connective tissue and is divided by occasional bony septa. They lose their coiled characteristic above the entering utricular nerve fibres and descend in bony canals toward the neuroepithelium. For the most part, they are separated from the nerve fibres which also traverse the bone. At the macula, the arterioles turn into the fibre layer to penetrate the loosely interlaced myelinated axons near the center of the neuroepithelial region.

The dense capillary bed found just below the basement membrane of the macula is limited to the hair cell area (see Figs. 3 and 4). It has the appearance of a flat, curved plate of radially directed, anastomosing vessels. The arterioles enter

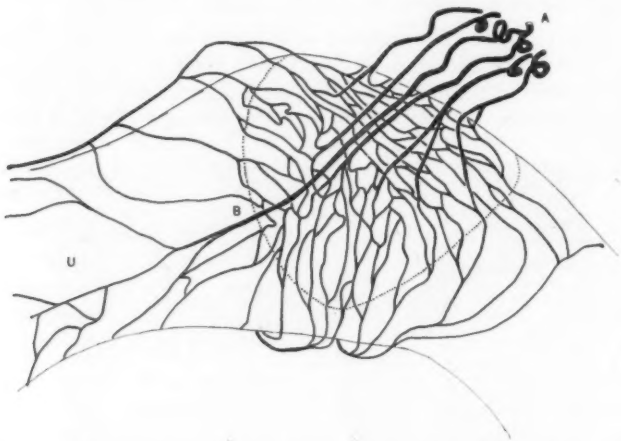

Fig. 3. Photograph of a schematic drawing showing the capillary ramifications of the macula of the utricle. The network is much simplified. The arterioles are shaded; the capillaries and venules are solid black. The limit of the hair cell region is indicated by the dotted line. (A) Arterioles coiling in the bone above utricular nerve; (B) arteriole entering loose network of utricle; (U) utricle.

near the center and the capillaries are brought together into venules at the periphery. Occasionally one of the arterioles enters the network at the edge and seems to be almost directly connected to a venule. Infrequently a venule has a central

connection. The collecting venules turn around the sides of the utricle, course in the connective tissue trabeculae and end in one of the large branches of the vestibular vein.

The remainder of the utricle is surrounded by a loose capillary network which is supplied by both main arteries. Some of the arterioles that originate in the coils above the macula bypass it and go directly to this utricular plexus. They cross the macula along the outside of the nerve fibre layer, just

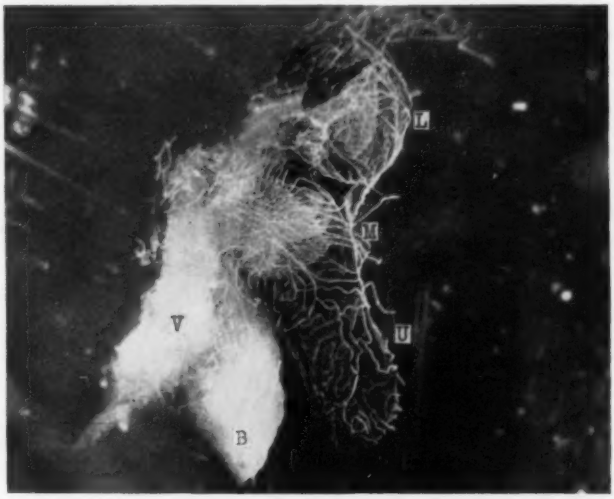

Fig. 4. Photomicrograph of a dissected specimen of the membranous labyrinth from guinea pig No. 102 showing the capillaries of the utricle and lateral ampulla. The superior ampulla can be seen underneath the lateral ampulla. The network of the medial attached surface of the utricle is down and less clear than that of the lateral surface. Intravascular precipitation of lead chromate; magnification 20×. (B) Bone; (L) lateral ampulla; (M) macula utriculi; (U) utricle; (V) vestibular nerve.

below the mesothelium and course to the edge of the neuroepithelium where they end in the loose utricular rete (see Figs. 2 and 3). The network is unevenly distributed on the free lateral surface. It appears to be more dense medially where the utricle is attached to the medial vestibular wall by connective tissue trabeculae.

On the lateral surface of the utricle the capillaries are found between the epithelial and mesothelial layers of the utricular wall. Medially, the vessels are free in the perilymphatic trabecular space. At the sides, the small collecting venules traverse this space and course toward the larger venous branches which are always closely related to the bone.

2. The Saccule. The macula acustica of the saccule receives its arterial supply from both anterior and posterior vestibular arteries. One or two of the coiled arterioles from the second branch of the anterior vestibular artery deviates toward the superior curved end of the saccule and enters the capillary network. The main arterial supply is derived from the posterior vestibular artery. These arterioles coil in the bone along the rim of the inferior wall of the vestibule, lateral to the macula (see Fig. 5). They straighten out as they turn

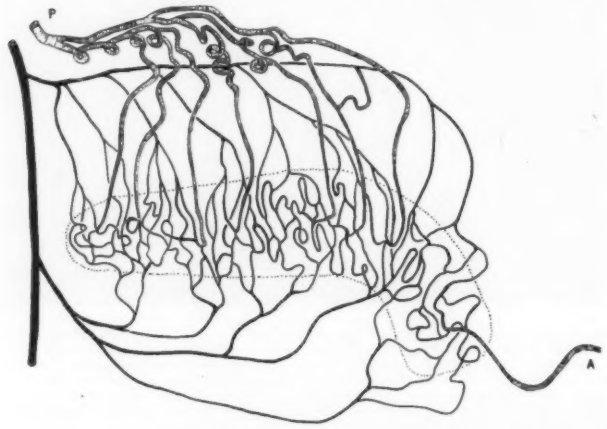

Fig. 5. Photograph of a schematic drawing showing the capillary ramifications of the macula of the saccule. The network is much simplified. The arterioles are shaded; the capillaries and veins are solid black. The limit of the hair cell region is indicated by the dotted line. (A) Branch from anterior vestibular artery; (P) branch from posterior vestibular artery.

toward the macula acustica, pass over the cribriform opening for the saccular nerve and enter the connective tissue with the myelinated fibres. The vessels continue in close proximity

to the periosteum toward the center of the macula. They turn toward the epithelium and pass in between the nerve fibres to enter the capillary network.

The subepithelial capillary bed is coextensive with the hair cells. The capillaries form a flat plate of loosely connected loops directed towards the edges of the macula and brought together into venules at its periphery. Two large veins, one anterior, the other posterior to the macula, receive the venous blood and course in thinly covered bony canals to the vestibular vein.

The free membranous covering of the saccule is generally avascular.

3. Ampullae of the Semicircular Canals. The three ampullae of the semicircular canals present a similar vascular pattern with some minor variations. That of the lateral canal will be described as typical. The terminal arterial branches make their final coils in bony channels apart from the ampular nerve. They divide and straighten out as they approach the ampulla. One large arteriole always passes over the side of the ampulla and ascends the bony semicircular canal. Two or three of the arteriolar branches enter the ampullary sulcus with the nerve fibres to supply the capillary network of the crista (see Fig. 6). Several branches of these do not enter the sulcus but go directly to the ampullary dome or beginning of the canal.

Near the midline of the crista, the arterioles enter the capillary network which is found between the epithelium and myelinated nerve fibres. The hair cells cover the densest part of the capillary network (see Fig. 7). At the border of the crista, some of the capillaries combine before crossing the planum semilunatum, but some may divide in the latter region. Near the outer border of the planum semilunatum they are joined by arterial branches and divide again before ascending the ampullary dome.

The capillaries of the ampullary dome are loosely adherent to the epithelium. They outline the membranous ampullary by

arching upward over it and at the ridge are directed toward the utricle (see Fig. 8). Along the ridge of the dome, a vein is formed by vessles from the proximal part of the semicircular canal and some of the arched vessels. At the utricle it has usually received the entire drainage of the ampulla and the proximal part of the semicircular canal.

Fig. 6. Photomicrograph of section through crista of the posterior ampulla showing the arteriole entering the crista and the subepithelial capillary plexus. (A) Arteriole entering crista; (S) subepithelial capillary plexus. (Na.) N. ampullaris.

4. Semicircular Canals. Each semicircular canal shows loosely connected capillaries accompanied by one or two large vessels throughout its entire length. At each end of the canal, a proximal plexus which is a continuation of that from the ampulla or utricle is found (see Fig. 8). These are drained by small venules that turn back toward the vestibule. The capillaries of the larger middle portion of the canal originate in a sizeable arterial branch from each end of the osseous canal. These follow the bone for the first millimeter or two

and only then approach the membranous canal to enter into the capillary network. A large vein draining the larger middle part exits from the utricular end of the canal. The posterior canal sometimes also shows a large vein leaving the ampullary end.

The capillaries do not completely encircle the canal. The larger vessels tend to course along its concavity with the capillaries ascending the sides between them. The convex sur-

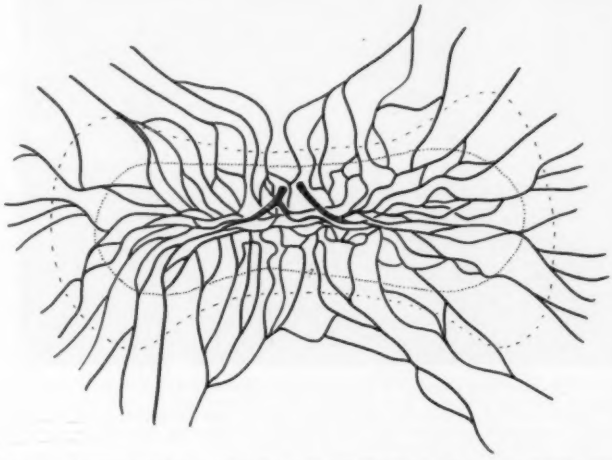

Fig. 7. Photograph of a schematic drawing showing the capillary ramifications of the crista. The epithelium has been flattened out, with the otolithic membrane downward. The network is much simplified. The arterioles are shaded; the capillaries are solid black. The limit of the hair cell area is indicated by the dotted line, that of the planum semilunatum by the broken line.

face of the canal which is closely applied to the bone is avascular. The vessels are found near the epithelium in the trabecular spaces.

5. The Nerve Fibres and the Vestibular Ganglion. The vestibular ganglion and the fibres of the superior ramus of the vestibular nerve are supplied by the common cochlear artery, dural vessels and supplementary branches from the anterior inferior cerebellar artery in addition to the vessels from the

anterior vestibular artery. It can be readily seen that the ganglion is more richly vascularized than the neighboring nerve fibres. The numerous capillaries intertwine among the ganglion cells with frequent anastomoses.

Fig. 8. Photomicrograph of a dissected specimen of the membranous labyrinth from guinea pig No. 102 showing the capillaries of the superior and lateral ampulla. Intravascular precipitation of lead chromate; magnification 20×. (A) Ampullary dome vein; (C) crista; (L) lateral ampulla; (L.C.) lateral canal; (S) superior ampulla; (U) utricle.

Further study is being made of the structure of the vessel walls and their innervation.

IV — Venous Drainage.

The venous drainage is generally accomplished by means of the vestibular vein (see Fig. 1). The confluence of veins draining the macula of the utricle and the superior and lateral ampullae gives it origin in the medial wall of the vestibule anterior to the utricle. It courses toward the round window in a shallow bony canal receiving small veins. As it turns toward the round window it skirts the edge of the saccule and receives the saccular veins, and a large vein from the

posterior ampulla, lateral canal and common crus, and the vestibular blind sac. The vestibular vein joins the veins from the cochlea and round window to form the vein of the cochlear aqueduct.

This appears to be the usual and main route of vascular exit, but there are always some vessels that leave the vestibule with the endolymphatic duct. One is of capillary size and probably arterial; the other is undoubtedly venous as it comes from the network of the under surface of the utricle and is often connected with the main vestibular vein. At times this vein is large and receives vessels from a considerable portion of the labyrinth. In some cases the large vein leaving the common crus was seen to exit in this manner. In other specimens a vessel receiving drainage from much of the utricle accompanied the duct. One ear was examined in which the vein was directly continuous with the vestibular vein, but was larger and evidently drained the entire vestibular labyrinth. This was unusual, and the opposite ear in the same animal showed no comparable vessel. All of these vessels empty into the sinus-like network which surrounds the endolymphatic sac.

The nerve fibres in the internal auditory meatus and those of the superior vestibular ramus in its bony canal appear to be entirely drained by the dural veins. Not only are the dural veins present in the internal auditory meatus, but they can be seen to continue upwards into the modiolus. In the previous studies[7] it was believed that the venules from the central nerve fibres in the modiolus passed into the posterior spiral vein. Further observation has indicated that the dural veins may also take part in the modiolar circulation of the guinea pig. They enter the modiolus by ascending close to the bone medial to the posterior spiral vein of the first turn and receive vessels from the bony capsule and possibly from the nerve fibres. The limit of their extent up beyond the basal turn cannot be determined. In consideration of the open character of the modiolus and the comparable situation of the superior vestibular ramus which seems to be completely drained by

dural veins, their possible relation to the central nerve fibres in the modiolus, and perhaps even an occasional connection with spiral ganglion cannot be disregarded.

DISCUSSION.

These observations, for the most part, confirm those made by Siebenmann,[1] Shambaugh[2] and Nabeya[5] regarding the course and distribution of the anterior and posterior vestibular arteries in the mammalian vestibule. All of these investigators have mentioned the variation in the origin of the anterior vestibular artery. Nabeya[5] has stated that in the guinea pig, as with other mammals, it was most often a branch of the labyrinthine artery. In the animals of this series it was found that the common cochlear artery and the anterior vestibular artery were usually separate branches of the anterior inferior cerebellar artery. The labyrinthine artery was seldom seen.

Siebenmann[1] clearly pictured the vessels of the membranous ampullae and Shambaugh[2] added the details in his description of the semicircular canals. There has been some disagreement as to the mode of drainage. Siebenmann[1] found a vein crossing the dome of the ampulla at right angles to the crista, draining the semicircular canal and ampulla. Shambaugh[2] observed a similar condition for the sheep, but found the vein coursed along the side in the domestic pig and calf. Nabeya[5] found conditions in the guinea pig similar to those in the human and the sheep. In these studies both methods of venous drainage have been observed. The ampullary dome vein was always found in the lateral and superior ampullae but not constantly in the posterior ampulla. Whether it courses over the ridge or along the side of the ampulla, it appears to receive drainage from only a small part of the canal; the larger part of the canal is drained by the large vein that usually is found leaving the utricular end of the canal.

The vascular pattern is similar in all of the neuroepithelial areas. The arterioles and nerve fibres enter the vestibule by separate bony channels, so that it is only in the ampullary

sulci, the spherical recess and just above the macula acustica of the utricle that vessels come into intimate contact with the vestibular nerve fibres. The arterioles enter the subepithelial capillary plexus near the center of the hair cell area, and the capillaries reach out toward the sides. The flow of blood is, therefore, from center to periphery. It was never observed in these animals that the arterioles and venules accompanied each other.

The dense plexuses are clearly limited to the hair cell areas. The form and shape of the capillary bed is adapted to the contour and size of the neuroepithelium. This is particularly clear in the two maculae. In the ampullae the situation is somewhat more complex. The more dense network is found under the crista, but many of the vessels do cross the planum semilunatum. The distribution of capillaries in the planum semilunatum is of interest in view of the attention that has been directed toward the possible secretory function[9] of the cells of this area.

In Nabeya's[5] mammalian studies, only the cat and guinea pig did not show the dual venous drainage of the vestibule that is present in other mammals and man. While he noted the presence of venous and arterial capillaries along the endolymphatic duct in the guinea pig, he considered them to be of little consequence. Experimental evidence for some collateral venous drainage has been presented recently by Perlman,[10] who found no sign of vestibular or cochlear dysfunction in the guinea pig after occlusion of the vein of the cochlear aqueduct. Observations in this series show that while the main route of vascular drainage from the labyrinth is through the vein of the cochlear aqueduct, the vein accompanying the endolymphatic duct is also of importance in the guinea pig. In some ears the vein leaving the vestibule with the endolymphatic duct was large and connected with a considerable part of the vestibular labyrinth. In one specimen the ramifications and size of the vein gave evidence that the venous outflow of the entire vestibular labyrinth was along the endolymphatic duct. In most, the vein was small and appeared to have no active part in the vascular drainage of the vestibule;

however, the loose subepithelial connective tissue in which it lies allows space for expansion, and it is probable that it is an ever present potential source of collateral drainage.

The capillaries in the trabecular spaces of the semicircular canals and under surface of the utricle have been mentioned by most investigators. The vessels which are found traversing these spaces filled in with connective tissue trabeculae are apparently bathed in perilymph. The importance of such a capillary relationship to fluid formation and resorption in the whole labyrinth is impressive; however, de Burlet[11] and Alexander[12] observed in certain mammals, including the guinea pig, that a membranous partition extends from the sides of the utricle and the ends of the semicircular canals to the osseous vestibule. The labyrinth is thus divided into a pars superior which includes the semicircular canals and under surface of the utricle, and a pars inferior. Evidence for the functional character of this partition can be seen in cross-sections of ears showing cochlear degeneration due to intense sound. There is often a heavy cochlear precipitate which extends into the vestibule, but it is limited to the anterior surface of the utricle and the saccule. This membranous partition separating one space filled only with fluid from another fluid space containing a loose web-like connective tissue traversed by small vessels may be compared to the situation found in the cochlea where a mesothelial covering separates the spiral ligament from the perilymphatic scalae.

The difference between the vascular-nerve fibre relationship in the vestibule and cochlea is worthy of comment. In the cochlea, the arteriolar vessels cross through the nerve fibres from vestibular to tympanic side in Rosenthal's canal and sometimes accompany the radial fibres from the spiral ganglion to the tunnel. Many of the venular capillaries also return along this route, turning back for the whole radial extent of Rosenthal's canal. In the vestibule the relation is less extensive. In the utricle, the capillaries contact the nerve only when they leave the bone and pierce the fibre layer to enter the capillary bed. In the saccule, although the precapillary arterioles are found in the connective tissue base of the

macula with the nerve fibres, they tend to remain close to the periosteum and pierce the fibre layer only before they enter the capillary bed. In the ampullae the arterioles enter the ampullary sulci with the nerve fibres. In neither utricle, saccule nor ampullae do the venules ever re-enter the fibre bundles, but always course away from the nerve.

SUMMARY.

1. The main features of arterial supply and venous drainage in the vestibular labyrinth as shown by previous investigators has been confirmed in these studies.

2. The most dense capillary networks are the subepithelial plexuses found below the hair cell areas.

3. The pattern of these plexuses below each neuroepithelial area is similar. The arterioles pierce the interlacing nerve fibres to form a flat, radial capillary network coextensive with the hair cells and are collected into venules at the sides. In the ampullae, diminished plexuses continue beneath the planum semilunatum.

4. The remainder of the membranous labyrinth is surrounded by a loose capillary network except along the convex surface of the semicircular canals and the free membranous covering of the saccule.

Acknowledgment is made to Dr. and Mrs. Wm. T. K. Bryan for their kindness in making the photomicrographs shown in Figs. 4 and 8.

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LATE DEVELOPMENT OF LARYNGEAL AND PHARYNGEAL CARCINOMA IN PREVIOUSLY IRRADIATED AREAS.

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Reports are appearing in the literature, of tumors arising in the larynx or hypopharynx following extensive irradiation of the neck for non-neoplastic diseases. While the total number of cases reported is as yet statistically insignificant, it appears that the latent period between the administration of therapy and the development of carcinoma is 20 to 35 years; since such therapy was most frequently advocated 25 to 30 years ago, similar cases may be seen with increasing frequency.

Six cases of carcinoma of the deep structures of the neck that occurred following irradiation of the cervical region were collected from the medical literature by Goolden[1] in 1951. Four were carcinomas of the laryngopharynx; one, of the larynx and one, of the thyroid gland. To this group, he added four cases observed in an 18-month period. All were females; they had received extensive irradiation of the cervical region 25 to 30 years before the occurrence of the tumor. In three of the cases, the irradiation was given for thyrotoxicosis, and in the last case, therapy was administered to tuberculous cervical lymph nodes.

To this group of 10 patients, three additional patients are

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The opinions expressed in this publication are not necessarily those of the Department of the Army, Department of the Navy, Department of the Air Force or Department of Defense.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Jan. 2, 1953.

added; each had received irradiation therapy for tuberculous cervical nodes and now has a malignant neoplasm of the hypopharynx or larynx in the direct line of the previously administered therapy.

CASE REPORTS.

Case 1: F. E., a 51-year-old white female, was first seen on July 1, 1949, complaining of sore throat and dysphagia; these symptoms had been present approximately one year. She stated that 32 years previously she was given a course of irradiation to the right side of the neck for a tuberculous infection of the cervical lymph nodes. Both the surgeon who referred the patient for treatment and the radiologist who administered the therapy are no longer living, and efforts to obtain the data regarding dosage, filtration, and other details have not been successful, since their records are no longer existent. Therapy was administered in the office of the radiologist and not in a hospital where records might have been more permanent. The patient states she has no recollection of the number of treatments or the length of each treatment but does recall that she received one treatment each week over the right side of the neck for a considerable period of time. While there was no apparent skin injury immediately following the treatments, this appeared later as a severe burn of the skin on the right side of the neck.

The general physical examination of the patient revealed a slight, white female, 51 years of age, with the only essential variations from normal associated with the pathology in the neck and pharynx. The skin over the right side of the neck showed the characteristic changes of extensive irradiation: atrophy, scarring, pigmentation and telangiectasis. The subcutaneous soft tissues appeared to be absent; the pulsating carotid artery, the lateral border of the thyroid and cricoid cartilages and the hyoid bone were readily visible and palpable immediately below the skin surface. Indirect laryngoscopic examination showed a partial fixation of the right vocal cord, edema of the right arytenoid and obliteration of the right pyriform sinus. The chest film demonstrated the heart to be of normal size and configuration and the lungs clear.

Direct laryngoscopic examination showed edema of all tissues in the region of the right pyriform sinus; a verrucous mass was present in the postcricoid area, and tissue was removed for histologic examination which revealed this to be a squamous cell carcinoma (see Fig. 1). Therapy consisted of irradiation of the tumor, using left lateral, anterior and posterior portals. A total tumor dose of 4,620 r. was given in a 37-day period. Following this treatment, the postcricoid tumor completely disappeared. Barium studies of the esophagus as well as esophagoscopy failed to reveal any pathology three months following completion of the treatment. The patient has been examined at three-month intervals since that time, and there has been no evidence of recurrence of the neoplasm in the three years following the establishment of the diagnosis.

Case 2: A. M., a 69-year-old white female, was first seen on May 22, 1950, because of hoarseness of three months' duration, loss of weight, dysphagia and pain in the right side of the throat. In 1935 she had had a hysterectomy for what reportedly was a malignancy and in 1941 a radical left mastectomy also reportedly for a malignancy. In 1920 she had multiple resections of tuberculous nodes on the right side of the neck, and this was followed by a considerable amount of X-ray therapy. Again, the dosage and other factors are not known but the skin changes were evidence that a large dosage was administered.

Mirror and direct examinations of the pharynx and larynx showed an ulcerative, necrotic mass involving the entire right side of the larynx, the right aryepiglottic fold, pyriform sinus and lateral pharyngeal wall. The left side of the larynx was edematous but not ulcerated. The biopsy

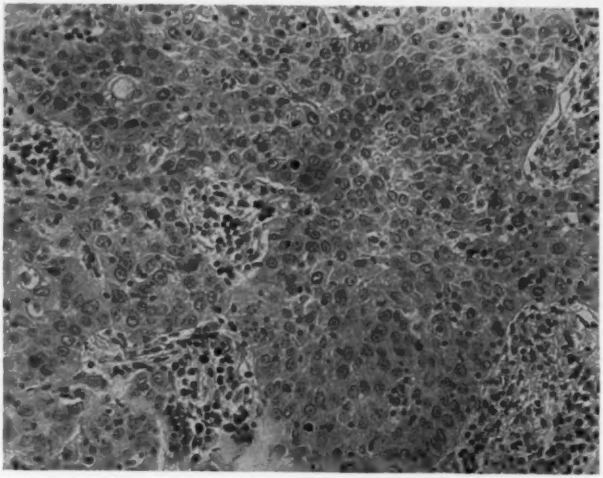

Fig. 1. Photomicrograph of biopsy of Case 1. Squamous cell carcinoma of the postcricoid space (×198).

demonstrated the tumor to be a squamous cell carcinoma (see Fig. 2). The entire right side was fixed. The lesion was considered inoperable, and external irradiation was administered from the left side as well as anteriorly and posteriorly, and subsequently 20 gold radon implants of 21 millicuries were injected into the residual tumor. A tracheotomy became necessary because of laryngeal obstruction, and while temporary improvement followed, her course was unfavorable and she expired on March 8, 1951, 10 months after the establishment of the diagnosis of the pharyngeal and laryngeal carcinoma.

Case 3: W. H., a 46-year-old Negro male, was first seen in February, 1952, complaining of difficulty in speaking and swallowing of about six weeks' duration. He had moderate and progressive dyspnea during this same period and had lost 18 pounds of weight. He stated that 27 years previously he had received a prolonged course of irradiation over the left side of the neck for a tuberculous infection of the cervical lymph nodes. During the course of treatment, he developed a severe burn of the skin on the left cervical region.

Examination of the patient revealed marked atrophic changes of the skin on the left side of the neck. Indirect laryngoscopy showed the presence of a large, firm mass completely filling the hypopharynx. Visualization of the larynx was obscured by the mass. There was no evidence of regional lymph node involvement. Biopsy of the mass was performed and histologic examination revealed it to be a fibrosarcoma.

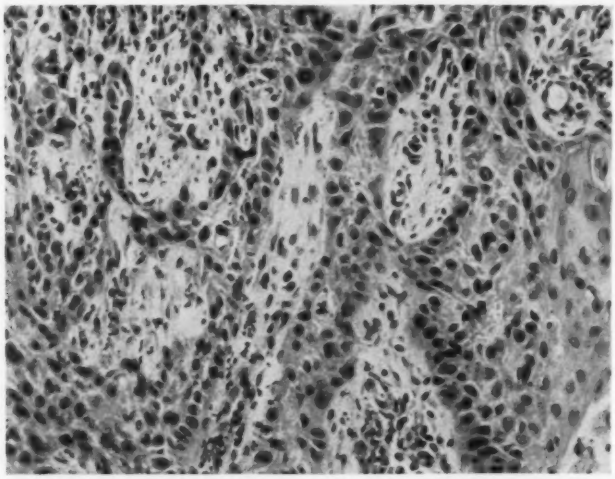

Fig. 2. Photomicrograph of biopsy of Case 2. Squamous cell carcinoma (×198).

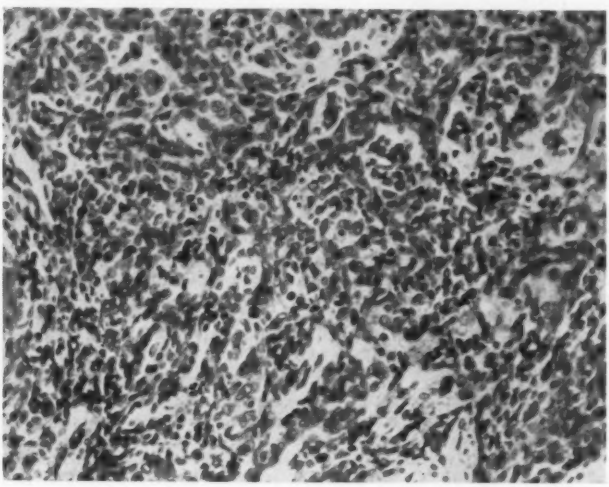

Fig. 3. Photomicrograph of tumor of Case 3. Fibrosarcoma of the larynx (×198).

On February 20, 1952, a right lateral pharyngotomy was performed. A firm, fairly smooth mass, measuring 5 cm. by 3 cm., was noted to fill the hypopharynx and supraglottic area completely. The mass was attached to the lateral aspect of the left aryepiglottic fold. It was resected along the area of attachment and the base was extensively dessicated with the electrodessicating current. The patient made a satisfactory postoperative recovery, and a tracheotomy tube, inserted at the time of operation, was removed. Histologic examination showed the mass to be a fibrosarcoma, the diagnosis obtained on biopsy (see Fig. 3). Examination six months after the operation revealed the involved area of the left aryepiglottic fold to have healed with no evidence of local recurrence or regional metastases.

DISCUSSION.

It is well recognized that carcinoma of the skin may develop in an area previously exposed to irradiation. Irradiation tumors developing in the deep tissues have been less thoroughly recorded and studied, possibly because they are less frequent, but also because they are less apparent and accessible. Hatcher[2] reported three cases and collected a total of 24 cases of sarcoma developing in previously irradiated bone. In 17 of the 24 cases collected from the literature the original condition treated was tuberculous arthritis. The interval between treatment and the appearance of the tumor was three to 12 years. Treatment factors were not always available, but in most cases it seemed excessive, and had consisted of repeated doses administered over a long period of time.

Smith and Bowden[3] reviewed 39 cases of carcinoma of the cervix developing in an area previously treated for a nonmalignant lesion by irradiation. The interval between therapy, and the onset of symptoms of the malignant condition varied from a minimum interval of 10 years to a maximal interval of 23 years.

Goolden[1] proposed certain criteria necessary to establish a diagnosis of irradiation carcinoma. These are as follows:

1. Microscopic evidence of malignancy.
2. Extensive irradiation administered for a disease not of neoplastic character.
3. A considerable lapse of time between the administration of the irradiation and the occurrence of the tumor.

4. The location of the tumor in the area of most concentrated exposure to the irradiation.

The three cases herein reported conform to these criteria and bring to 12 the number of postirradiation malignancies of the pharynx and larynx reported in the literature. One patient, No. 3 of Table 1, is included in Goolden's review of cases although it is not actually a case of carcinoma of the larynx or hypopharynx. It is an adenocarcinoma of the left lobe of the thyroid that developed immediately below the

TABLE I

Pt.	Age	Sex	Original condition treated	Interval between irradiation & malignancy	Anatomical site	Pathology	Author
1		M	Tuberculous cervical adenitis Treated from 1919 to 1922	13-16 Therapy given for 3 years	Carcinoma of the larynx	Squamous cell carcinoma	Lesser[4]
2	65	F	Goiter	20 years	Carcinoma of the posterior wall of hypopharynx	"Carcinoma Simplex" of hypopharynx	Kruchen[5]
3	54	M	Tuberculous cervical adenitis	24 years	Adenocarcinoma of left lobe of thyroid. Post-irradiation atrophy and scarring in skin and subcutaneous tissues overlying carcinoma	Adenocarcinoma of thyroid	Kindler[6]
4	59	F	Thyrotoxicosis	20 years	Tumor surrounding pharynx at post-cricoid level	Squamous cell carcinoma	Kindler[6]
5	43	M	Tuberculous cervical adenitis	27 years	Aryepiglottic fold. Necrotic ulcer in irradiated area of skin	Carcinoma of the larynx	Den Hoed[7]
6	41	F	Tuberculous cervical adenitis	28 years	Aryepiglottic fold and arytenoid cartilages. (3 years previously basal cell ca in irradiated area of skin excised)	Squamous cell carcinoma	Den Hoed[7]
7	69	F	Thyrotoxicosis	26 years	Mass involving left pyriform sinus and left aryepiglottic fold	Squamous cell carcinoma	Goolden[1]
8	50	F	Thyrotoxicosis	29 years	Mass involving post-cricoid area and aryepiglottic fold and extending below cricopharyngeus	Fibrosarcoma	Goolden[1]

atrophic, scarred skin and subcutaneous tissues that had received irradiation for tuberculous cervical lymph nodes 24 years previously. It thus falls into the category of irradiation carcinoma of a deep structure of the neck.

It cannot be merely coincidental that all cases reported had received irradiation 16 to 32 years before the development of

the neoplasm. In the three cases herein reported, the tumor had its origin on the side previously irradiated. The sex distribution of the 12 cases, nine females, three males, may be significant, but the group is too small to draw any definite conclusion; however, since the pyriform sinus and the laryngeal lesions are seen most commonly in males and only the postcricoid lesions most frequently in females, this sex distribution may be of interest.

TABLE I CONTINUED

Pt.	Age	Sex	Original Condition Treated	Interval between Irradiation & Malignancy	Anatomical Site	Pathology	Author
9	76	F	Thyrotoxicosis	27 years	Mass in post-cricoid area. Deep cervical lymph node extension	Squamous cell carcinoma	Goulden
10	49	F	Tuberculous cervical adenitis	30 years	Mass involving posterior wall of hypopharynx, post cricoid area and cricopharyngeus.	Squamous cell carcinoma	Goulden
11 F. P.	51	F	Tuberculous cervical adenitis	30 years	Mass in post cricoid area and right pyriform sinus. Extensive burn of skin of right side of neck.	Squamous cell carcinoma	Holinger and Rabbett
12 A. M.	69	F	Tuberculous cervical adenitis	30 years	Extensive, ulcerating, destructive mass involving entire right side of pharynx and larynx.	Squamous cell carcinoma	Holinger and Rabbett
13 M. H.	46	M	Tuberculous cervical adenitis	27 years	Large pedunculated mass filling supraglottic space and hypopharynx arising from lateral border of left aryepiglottic fold. extensive burn of skin on left side of neck.	Fibrosarcoma	Holinger and Rabbett

Of the 12 patients, seven received treatment for tuberculous cervical adenitis and five for thyrotoxicosis. Histologic proof of the diagnosis of the original lesion is not available. As stated above, Hatcher[2] collected 24 cases of sarcoma developing in previously irradiated bone; in 17 of these the original condition treated was tuberculous arthritis. In the 39 cases of carcinoma of the cervix developing in a previously irradiated area reported by Smith and Bowden,[3] the conclusion that the lesion previously treated was benign was based on a negative original biopsy or the fact that an asymptomatic interval of 10 or more years elapsed between the irradiation and the onset of symptoms of the malignancy.

SUMMARY AND CONCLUSIONS.

Three cases of malignancy of the pharynx and larynx are reported in which the tumor developed in an area previously irradiated for a benign process. A review of the literature reveals the reports of nine similar cases; in the total series of 12 cases, there were three males, nine females. All had received irradiation 16 to 32 years before the development of the neoplasm; seven received irradiation for tuberculous cervical adenitis and five for thyrotoxicosis. Cases of carcinoma of the skin developing in a previously irradiated area are well known. Recently, malignancies have been reported developing in deeper structures previously treated for benign lesions. This has occurred in bones as in the development of bone sarcomas in areas treated three to 12 years previously for tuberculous arthritis, and carcinoma of the cervix developing 10 or more years after irradiation treatment for a benign lesion of the cervix. While statistically the series of 12 pharyngeal and laryngeal malignancies is small, it would be difficult to believe that the association of the irradiation and the development of the tumor is merely coincidental. Since irradiation therapy was more common for benign conditions of the neck several decades ago, more of these cases may be seen in the near future.

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ENDAURAL MODIFIED RADICAL MASTOIDECTOMY.*

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In many selected cases of chronic otorrhea, the radical mastoid operation may be avoided and good serviceable hearing preserved or the hearing even improved by doing a modified radical operation. In my opinion, it is in the various modifications of the radical mastoidectomy that the endaural approach offers the most promise for advances in the treatment of chronic otorrhea, secondary to disease of the mastoid, mastoid antrum or attic areas.

Since 1938, when Lempert[1a] reported on his endaural approach to the temporal bone, numerous articles have been written on the use of this approach in the treatment of all types of diseases of the temporal bone.

Lempert,[1] Meltzer,[2] Shambaugh,[3] Woodruff and Henner,[4] House,[5] Maxwell,[6] Baron[7] and others have pointed out its usefulness in carrying out the modified radical mastoidectomy and have advocated the modified radical operation in certain types of chronic ears; however, the importance and value of the modified radical mastoidectomy have not been widely taught or adopted.

In the past, the modified or conservative mastoid operation has been associated with such names as Jansen,[8] Körner,[9] Bárány,[10] Heath,[11] Bondy,[12] Siebenmann,[13] Ballenger,[14] Richards[15] and others.

The so-called conservative mastoid operation was first introduced in this country by Heath,[11] in his article published

*Read at the meeting of the Eastern Section, American Laryngological, Rhinological and Otological Society, Inc., Syracuse, N. Y., Jan. 7, 1953.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Jan. 9, 1953.

in *The Lancet* in 1906. In 1911, Richards[15] gave a detailed description and comparison of the modifications of the radical operation as done by Heath, Bondy and Siebenmann.

The Siebenmann[13] operation differed from the classical radical operation somewhat, in that Siebenmann limited the scope of the operation to the removal of only so much of the bone of the mastoid region and attic area as was necessary to make all parts accessible through the external canal. The lateral attic wall, if touched, was only partly removed. The drum, malleus and incus were *removed.* An essential part of his operation was the "Y" type of incision for the flap, which is known as the Siebenmann flap.

Heath,[11] in his conservative mastoid operation, used the postauricular incision but meticulously preserved the membranocutaneous canal wall. After removing the posterior and superior bony canal wall, he cut his original plastic meatal flap, which was somewhat similar to the Stacke[16] plastic flap. The flap was then retracted and the tympanic area inspected. By means of a rubber bulb and attached catheter, air was passed from the aditus through the attic and/or the middle ear, depending upon the perforation. Finally, in similar manner, a lotion was forced through the attic. If cholesteatomatous material appeared in the washings, Heath elected to do the radical operation. If there was no evidence of cholesteatoma, and tympanic conditions indicated the possibility of repair, Heath proceeded with his conservative operation. It is possible that Heath did his conservative operation in certain cases where a simple mastoidectomy would have sufficed.

Bondy[12] did his conservative operation by beginning with the simple mastoidectomy technique. After the facial ridge was taken down by the chisel, Bondy completely removed the lateral attic wall and made the bridge as thin as possible, but did not remove it. The usual plastic canal flap was cut after the bone work had been completed. Bondy elected to remove the body of the incus and head of the hammer later under local anesthesia, if purulent discharge continued, due to involvement of these structures.

In the conservative operations of Heath and Siebenmann, the lateral attic wall either was left untouched or a part was allowed to remain for the insertion of the tympanic membrane. The bony external canal was always maintained until last to avoid dislocation of the ossicles.

Fig. 1. X-rays of mastoids (Law's position). Left—showing gross destruction (Case 4). Right—showing sclerosis with some destruction (Case 6).

Fig. 2. X-ray of mastoids (fronto-occipital view). Left—marked periantral destruction (cholesteatoma) (Case 4). Right—moderate periantral destruction (cholesteatoma) (Case 6).

The conservative operation of Bondy differed from the Heath operation in a number of fundamentals: Bondy removed the lateral wall of the attic, thus exteriorizing this area for inspection and treatment. Here the bridge was made as thin as possible but was not removed, even though Bondy felt that the drum did not need a bony support since there was adequate support given by the ligaments in the region of the Rivinian fissure. It is also interesting to note that Bondy did not consider Shrapnell's membrane as a part of the tympanic membrane proper.

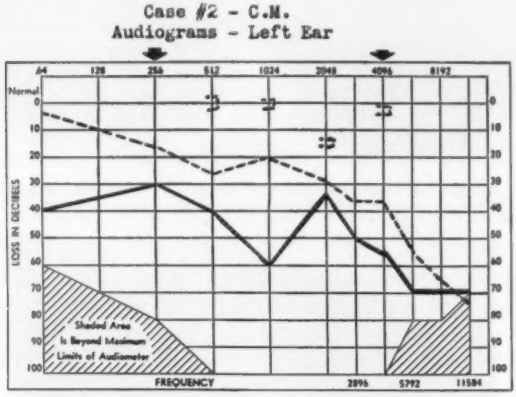


Fig. 3. Composite pre- and postoperative audiograms, Case 2 (C. M.).

Bondy used his operation only in chronic cases and included cases of cholesteatoma. He doubted that there was danger from recurrence of the cholesteatoma after removing the lateral attic wall, as the cholesteatoma was completely removed.

The indications for the Bondy conservative mastoid operation, as quoted by Richards,[15] are as follows: "A hearing distance, before the operation, of two meters or more, provided it is probable that there is continuity of the ossicular chain and that the middle ear cavity is itself not diseased, or

but slightly. It is especially indicated where the perforation is limited to Shrapnell's membrane, the drum membrane intact, and the hearing good, provided there is no material improvement after a period of conservative treatment of four weeks."

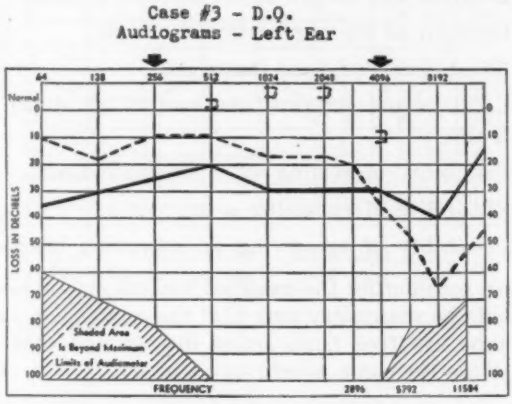

Case #3 - D.Q.
Audiograms - Left Ear
LOSS IN DECIBELS
FREQUENCY
Shaded Area Is Beyond Maximum Limits of Audiometer
5/6/48 - Pre-Operative - 14.2% loss——
7/24/52 - 4 yrs.Post-Op. - 2.3% loss----
Date of Operation - 5/7/48

Fig. 4. Composite pre- and postoperative audiograms, Case 3 (D. O.).

In recent years, interest in the modified radical operation has been revived, largely as the result of the development of Lempert's[1] tympanomeatal flap and all its various modifications by those who have explored the possibilities of the endaural approach.

The tympanomeatal flap was first originated by Lempert[1b] for use in the modified radical or mastoidoatticotomy and later was used as the foundation for his one-stage fenestration operation.

INDICATIONS.

In considering the indications for the modified radical mastoid operation, the following factors are involved:

1. Condition of the middle ear and Eustachian tube.

2. Condition of the pars tensa of the tympanic membrane.
3. Degree of hearing loss in the affected ear.
4. Degree of hearing loss in the opposite ear.
5. Location and nature of the diseased process.
6. Location of the perforation or fistula.
7. Effectiveness of local treatment.
8. Location and extent of destruction, as shown by X-ray.
9. Possibility of sealing off of the middle ear.
10. Possibility of obtaining a dry ear.
11. Possibility of preserving or improving hearing.

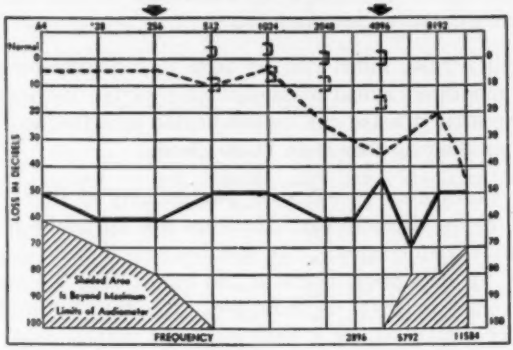
Before considering the modified radical mastoid operation, one should be reasonably sure that the middle ear and Eustachian tube are free from active disease and that the pars tensa of the tympanic membrane is intact.

Under these conditions, the ideal case for the modified radical operation is one with fair to good hearing and with the diseased process limited to the mastoid antrum or epitympanic space, with either an attic perforation or an antral fistula through the posterior superior bony canal wall.

For practical purposes, the commonest type of case suitable for the modified radical operation is the one with cholesteatoma formation involving the mastoid antrum, with or without invasion of the attic region. Here the perforation is usually in Shrapnell's membrane, or else is marginal, involving the lateral wall of the attic. Frequently the middle ear is entirely sealed off by the cholesteatoma matrix and is functionally normal.

Occasionally one encounters a large posterior attic perforation with a relatively small cholesteatoma in the mastoid antrum or posterior attic area, which can be treated adequately by conservative methods; however, if there is involvement of the attic area proper, particularly with a small

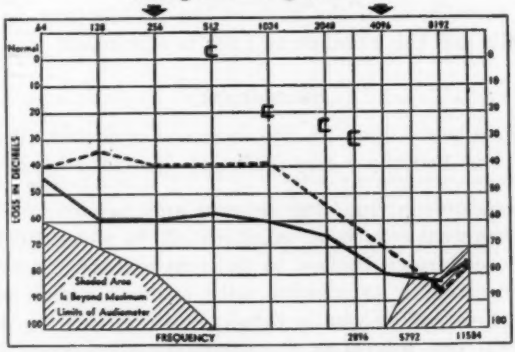
Case #4 - P.D.
Audiograms - Left Ear



11/11/48 - Pre-Operative - 54.0% loss ——-
3/20/51 - 2½ yrs.Post-Op.- 4.3% loss ----
Date of Operation - 11/12/48

Fig. 5. Composite pre- and postoperative audiograms, Case 4 (P. D.).

Case #12 - A.L.
Audiograms - Right Ear



3/20/51 - Pre-Operative - 69.4% loss ——
10/6/52 - 1½ yrs.Post-Op. - 43.3% loss ----
Date of Operation - 3/20/51

Fig. 6. Composite pre- and postoperative audiograms, Case 12 (A. L.).

perforation, then conservative measures are almost sure to fail, resulting in eventual involvement of the middle ear and progressive hearing impairment.

In such a case, much can be done to conserve hearing by doing a modified radical mastoidectomy early. By such treatment, the attic disease can be eradicated and frequently with no greater hearing loss than 20 to 25 db.

Other cases may be suitable, particularly if there is poor hearing in the opposite ear. Here we are apt to be more liberal in our requirements. If the hearing in the ear to be operated upon is poor, but still the better ear, the conservative operation may yet be indicated, providing the middle ear is already sealed off by a cholesteatoma matrix and free of active disease.

Frequently the advisability of doing a modified radical operation depends upon the possibility of obtaining a middle ear which is sealed off from the exteriorized postoperative cavity. This is true because, if the middle ear is already sealed off or can be sealed off at operation or in the postoperative period, one is virtually assured of a dry ear, since the possibility of continued drainage from secondarily infected mucous membrane in the area of the promontory, hypotympanic cells and the Eustachian tube is eliminated.

TECHNIQUE.

All cases were done by the endaural approach, using the incision and technique essentially as described by Lempert.[1]

The mastoid antrum was entered and any remaining diseased periantral cells were exenterated. In several cases, the disease was largely limited to the mastoid antrum area and posterior attic, in association with either a small posterior attic perforation or else a fistula in the posterior superior canal wall, just lateral to the tympanic membrane. In such cases, the aditus was enlarged and the mastoid aspect of the posterior and superior canal walls skeletonized and removed, taking care to leave the membranous canal wall intact. In

only two cases was I satisfied, with the aid of magnification and a mastoid searcher, that the epitympanic spaces, middle ear and the ossicles were free from disease (Cases 4 and 6).

Fig. 7.

SUMMARY CHART — MODIFIED RADICAL MASTOIDECTOMIES — 18 Cases

Case No.	Hearing: Improved Unchanged Worse	Improvement in %	Increased Loss in %	M.E. Involved Pre-Op.	Incus Left	Head of Malleus Left	Pars Tensa Intact & M.E. Sealed Off	Dry Ear
1.	Worse	-	12.9%	Yes	No	Yes	No	No
2.	Improved	27.6%	-	No	No	No	Yes	Yes
3.	Improved	11.9%	-	No	Yes	Yes	Yes	No
4.	Improved (aural polyp)	40%	-	No	Yes	Yes	Yes	Yes (Heath)
5.	Improved	10.1%	-	Yes	Yes	Yes	Yes	Yes
6.	No change	-	-	No	Yes	Yes	Yes	Yes (Heath)
7.	Worse (sl.)	-	6.7%	No	Yes	Yes	Yes	Yes
8.	Worse (sl.)	-	10.6%	Yes	Yes	Yes	No	No
9.	No change	-	-	No	Yes	Yes	Yes	Yes
10.	Worse	-	12.9%	Yes	No	No	No	Yes
11.	Worse	-	28.6%	Yes	Yes	Yes	Yes	Yes
12.	Improved	26.1%	-	No	No	No	Yes	Yes
13.	No change	-	-	No	No	No	Yes	Yes
14.	No change	-	-	No	No	No	Yes	Yes
15.	No change	-	-	No	No	No	Yes	Yes
16.	No follow-up audiogram			No	Yes	Yes	Yes	Yes
17.	Improved	21.7%	-	No	Yes	Yes	Yes	Yes
18.	Improved	15.9%	-	No	Yes	Yes	Yes	Yes

Dry Ears: Yes - 15 .. 83%
No - 3

Hearing: Improved 7) 12 or 66%
No essential change 5)
Worse 5
Lost to follow-up 1

Average Improvement in % ... 21.9%

Average increased loss in % .. 14.3%

Hearing Worse:
1. Residual M.E. infection and involvement of head of the malleus.
8. Slight residual infection in attic.
10. Anterior superior perforation of M.T. M.E. not sealed off above perforation.
11. No reason apparent for progressive loss.

In these two cases, the Heath-type[11] procedure was carried out, leaving the lateral attic wall intact for the most part, and the ossicles undisturbed. A cutaneous canal flap was cut and reflected back over the facial ridge, as in the original Heath procedure.

I am convinced that it is extremely important to do the bone work necessary to explore the mastoid antrum, aditus and epitympanic space at the same time, keeping the membranous canal wall intact before deciding whether or not to do a modification of the radical, or to do the usual radical mastoidectomy.

If the epitympanic space is involved, the entire lateral attic wall is resected, and, under magnification, all diseased tissue is removed. This removal is aided by a dry field, through the use of adrenalin packs, irrigation with normal saline solution, and the use of a small Frazier suction tip.

After all pathological tissue, including cholesteatoma and granulations, has been removed, the ossicles are inspected. If satisfied that they are normal and not involved by necrosis or granulations, they are left undisturbed. If involved, the incus and head of the malleus are removed. If the incus is necrotic and has to be removed or has sloughed, the head of the malleus should also be removed. By doing this, one avoids considerable crusting, and the middle ear is more easily sealed off by a plastic typanomeatal flap.

Cholesteatoma Matrix: For years there has been considerable discussion as to the advisability of complete removal of the cholesteatoma matrix versus the preservation of at least some of the matrix to hasten healing. Baron[7] has written an excellent article on the "Modified Radical Mastoidectomy," in which he goes into a detailed discussion of the various types of cholesteatoma and the pros and cons regarding the removal or preservation of the cholesteatoma matrix. He urged the preservation and use of the matrix to shorten the period of epidermization.

If cholesteatoma invades the attic area, it is essential that the lateral attic wall be taken down and any cholesteatoma or granulation tissue be removed; however, if the middle ear appears healthy and is completely sealed off by a cholesteatoma matrix, it is not necessary to remove this covering matrix so long as the cavity containing the matrix is exteriorized and made accessible. The presence of the cholesteatoma

matrix considerably shortens the time required for complete epidermization. The postoperative cavity is ultimately lined with the same type of squamous epithelium as the cholesteatoma matrix, and, if there are not underlying pockets of infection or disease, I see no reason to prolong the period of epidermization or to take additional time for more extensive use of skin grafting.

Depending upon what is found in the epitympanic space, several choices of procedure are available:

1. If the ossicles are left, they may be
   a. covered over by a tympanomeatal flap as described by Lempert,[1b] or
   b. left exteriorized, as described by Meltzer.[2]
2. If the ossicles are covered over by a fairly healthy cholesteatoma matrix and the middle ear is sealed off, with the pars tensa intact, I have left them undisturbed, with good results.
3. If the ossicles are removed, then the middle ear may be sealed off by
   a. a tympanomeatal flap, or
   b. left to seal itself off, as it will not infrequently do. (Occasionally one will find the incus and head of the malleus destroyed by disease and missing, with the pars tensa intact and the middle ear sealed off by cholesteatoma matrix. Some of these cases have good hearing, and I feel the middle ear can be left alone—Case 2.)
4. If the tympanomeatal flap is not used, then a canal flap is cut, as described by Shambaugh,[3b] and turned back over the facial ridge.
5. If a fistula is present in the horizontal semicircular canal, as it frequently is, this can be covered over by a type of tympanomeatal flap or by a small full-thick-

ness free graft. Any cholesteatoma matrix over the fistula is removed, but the granulations over the membranous semicircular canal are not disturbed.

Multiple small full-thickness grafts from the excised conchal skin are useful to line key points to hasten healing.

The cavity is packed with paraffin-meshed gauze, containing aureomycin ointment, and the usual dry sterile dressing is applied.

POSTOPERATIVE CARE.

Postoperatively, 400,000 units of penicillin every eight hours are used until some time after the packing is removed. If Gram-negative organisms, such as Bacillus Pyocyaneus, are present, streptomycin or a suitable antibiotic is added. The packing is removed on the sixth day. The cavity is inspected and cleaned daily, using sterile technique. Packing is used to control the size of the meatal opening. Granulations, if they occur, and they usually do if much infection is present, should be removed with a sharp ring curette. Local application of gentian violet is useful to keep down infection. Ninety-five per cent alcohol with 2 per cent boric acid, as ear drops, twice a day, is useful in stubborn cases. Aerosporin Solution (put out by Burroughs, Wellcome and Co.) , used by local application or as ear drops, is the most effective agent against Bacillus Pyocyaneus that I have found. With the use of antibiotics immediately postoperatively and aureomycin packing, it is rare that a free graft has to be removed or sloughs.

SUMMARY.

Eighteen cases of the modified radical mastoidectomy are presented. All cases had intermittent otorrhea for one or more years, and the average duration of the discharge was 13 years. All cases, except one, showed evidence of cholesteatoma at the time of operation.

The Heath type operation was done on two ideal ears, with excellent results. Postoperative healing and epidermization were unusually rapid, resulting in dry ears. After over four

years' observation in one and a little under four years in the second, the ears have remained dry and the hearing excellent. There is no per cent loss in one ear, and only 4.3 per cent loss in the other.

In the remaining 16 cases, the Bondy type operation was done. In this group, the results varied considerably according to the pathology and the method of treatment.

In the entire series, dry ears were obtained in 15 of the 18 cases, or 83 per cent. The hearing was improved in seven; there was no essential change in five; there was additional hearing loss in five; and in one case there was no follow-up audiogram. The average time required for complete epithelization and to achieve a dry ear was 10 weeks.

In cases with improvement in hearing, the average improvement in per cent was 21.9 per cent; and in the cases with further loss, the average increased loss in per cent was 14.3 per cent.

The five cases which showed increased loss were reviewed critically to determine, if possible, why there was additional loss. In at least three of the five cases, there was evidence of incomplete removal of the diseased tissue from the attic area and/or involvement of the middle ear. In two of the five cases, a dry ear was not claimed, although at present Case 1 shows only slight crusting in the anterior attic area.

CONCLUSIONS.

In the treatment of chronic suppurative ear disease, secondary to infection of the mastoid, mastoid antrum or attic areas, one should endeavor not only to treat adequately the diseased process, but also, wherever possible, to maintain or even improve the hearing.

The choice between the radical and the modified radical mastoid operation should be made only at the operating table, after thorough inspection and study of the tympanic and attic areas, under magnification with full exposure and adequate illumination.

The choice of operation should be made with the patient's safety as the first objective; however, in many cases of chronic ear disease, the infection can be dealt with adequately, a dry ear obtained, and, at the same time, the hearing can be preserved or even improved by the modified or conservative operation.

The technical procedure can be accomplished by either the classical postauricular route or endaurally. In my experience, the endaural approach, aided by magnification and a dry surgical field, adds to the safety and effectiveness of the modified radical mastoid operation.

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ADVANCES IN THE USE OF ANTIBACTERIAL SUBSTANCES.*

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During the past decade, many surgical procedures in the regions of the ear, nose and throat have become almost extinct. There is no question that the wise prescription of antibacterial substances for infections in these areas has reduced impressively the indications for the drainage of infections in the middle ear, for mastoidectomies, radical operations upon the sinuses, tracheotomies and for a variety of other surgical treatments; furthermore, local and distant purulent complications of infections arising in the upper respiratory passages are appreciably less common now than 10 years ago. These facts prove that antibacterial substances have indeed lessened serious infectious disease in mankind.

The physician now has easily available at least 10 antibacterial substances which are safe to use locally or orally; a few are dangerous when administered parenterally: penicillin, the sulfonamides, streptomycin, terramycin, aureomycin, chloramphenicol, bacitracin, polymyxin, gramicidin and/or tyrothricin, and para-amino-salicylic acid. During the past 12 months, three new agents have been added to this list and within the next two or three months at least another two will be available: isoniazid, stilbamidine and erythromycin have been accepted by the F. D. A. and magnamycin and thiocymetin shortly will be available for distribution. These five drugs deserve some comment. Three, erythromycin, magnamycin and thiocymetin are in general broad spectrum agents, active against a reasonably wide range of bacteria. Erythromycin and magnamycin are best used in infections caused by Gram-

*Read at the meeting of the Eastern Section, American Laryngological, Rhinological and Otological Society, Inc., Syracuse N. Y., Jan. 7, 1953.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Jan. 9, 1953.

positive organisms and are, therefore, quite comparable to aureomycin. Each probably is no more active than either aureomycin or terramycin, and dosages are of the same order. In addition, first experiences with these new agents suggest that each antagonizes organisms resistant to penicillin. Thiocymetin is active primarily against Gram-negative organisms and so is somewhat similar to both terramycin and chloramphenicol; however, to date, too few experiences have been recorded with all of them for comparison with any other drug.

Whether or not they will prove valuable, to the physician is unimportant; but I would stress one feature of their production which is very important, namely, they are representative of the work which indicates that advances in the therapy of infections are continuing rapidly and effectively. These advances represent great expenditure of money and human resources on the part of every drug manufacturer, without government subsidy. Industry, fortunately, is constantly searching for new and better antimicrobial agents. I have the greatest confidence in and respect for this sincere effort on the part of private industry to advance knowledge by producing new drugs. It will be a sad day indeed when manufacturers refuse to try further to discover the ideal. Each new agent as it comes out must be given a fair trial before being discarded to encourage the manufacturers to proceed, and, because each new material represents an advance, perhaps small, in our overall knowledge of bacterial antagonism and host-drug-parasite relationships.

In the past year two very impressive advances have been made: *Stilbamidine* is a chemical which in the past 10 years has been used in a variety of diseases, such as multiple myeloma, and has never quite become popular; however, by chance it was discovered to have great activity against *blastomycoses*. Now the formerly indolently chronic and fatally progressive infestation can be well controlled and probably cured by a fungicide. The activity of stilbamidine does not extend to most species of actinomycoses nor to the dermatophytins and other fungi, but its usefulness in blastomycotic infections

shows that it is the first drug to be administered parenterally, which antagonizes the growth of fungi in the human. This advance may be of minor import to otorhinologists because certainly it will be used very infrequently, since the infection, fortunately, is rare; but to those of us in the field of infections the advance is spectacular and one of the most encouraging discoveries since penicillin and streptomycin.

The second signal advance has been in the field of tuberculosis. Derivatives of isonicotinic acid hydrazide, not a drug of biologic origin, have been shown to exert a bacteriocidal effect upon the growth of tubercle bacilli of an order similar to that of streptomycin, and in some respects, an even greater effect. The drug is cheap, completely innocuous and active in the human. Unfortunately, like streptomycin, tubercle bacilli become resistant to the chemical after short exposures of approximately 90 days, and as a single drug in the disease, it is, therefore, not likely to succeed; however, when combined with streptomycin its high antituberculous activity can be prolonged and this combination of drugs produces the best results ever observed in the long term management of tuberculosis in any body tissue or system. There are two reasons why its discovery is so very important: first, it actually represents the first chemical since the sulfonamides to have more than mere bacteriostatic properties. No longer is it necessary to search for agents with bactericidal properties only from biologic origins. Isoniazid also represents the first agent, cheap and easy to take, which might allow tuberculosis to be treated on an ambulatory basis.

These are the recent major advances in the use of antibacterial substances. There are many others, mostly mechanistic, some are sensible and three will be discussed: neopenil, bicillin and combinations of various agents in a single carrying material.

Neopenil is penicillin G diethyl amino ethyl ester hydroiodide. It is an insoluble iodide salt of penicillin, has similar antibacterial activity as penicillin but has two added pharmacologic properties which allow it some advantage. It is

singularly unique in that it is the first preparation of penicillin to penetrate the blood brain barrier. It is, therefore, a valuable agent in the management of meningitis. Standard doses of the material result in concentrations approximately tenfold greater in spinal fluid than those expected after the administration of soluble penicillin G. This property should make even more secure the results of therapy of both pneumococcic and meningococcic meningitis — both central nervous system infections frequently associated with foci in upper respiratory areas. Its second and far less important property is that it is found in significant concentrations in bronchial secretions. Unlike the advertising, however, this advantage is not really very important because the drug offers no better therapy for tracheobronchial infections than the regular forms of penicillin.

Bicillin is another ester of penicillin G, dibenzyl ethylene diamine di penicillin. It is almost completely insoluble in body fluids and is, therefore, a material which upon injection is released at an extraordinarily slow rate and in very low concentrations. One dose of 300,000 to 600,000 units will result in blood levels extending well over seven days. It has very little place in the therapy of active infections but does have a very important place in any long term program for prophylaxis.

The use of two or more antimicrobial substances together deserves serious comment for various reasons. Although not necessarily to be wholly condemned, the practice of prescribing two or more drugs together is associated with certain dangers, but, more important, *no* real advantage results. It must be emphasized that to achieve success in the management of any infection, the organism must be exposed to sufficient concentrations of its antagonist. If that occurs, no added amounts of the agent or any other in combination with it will do a better job of killing the organism — or, more often, of preventing its reproduction. If the concentration of the agent is not sufficient, then its administration is faulty and deserves revision, or the wrong agent is being used. It

most assuredly is true that two agents or more will do no better than one alone, if the one is used wisely. There are very few exceptions to this general rule. An antimicrobial agent acts only upon the infecting microorganism — it has no power of healing an area of inflammation, nor actually is it capable of eradicating all of the organisms. It has never been shown, for example, that mixture of bacitracin and polymyxin, or of penicillin and sulfonamide, or of any other combination are more specific than the better of the two drugs alone, in the management of a single infection.

There are, however, exceptions to this general rule: in infections caused by organisms partially resistant to a single drug, on occasion the clinical response will be more impressive with two drugs together. When an infection is caused by more than one micro-organism, and particularly if the mixture of organisms contains both Gram-positive and Gram-negative forms, then combinations of drugs are in order; but this actually occurs quite uncommonly. Despite controversial literature upon the subject, polybacterial infections in the respiratory passages are almost rare; therefore, the vast majority of infections do not deserve polyantimicrobic therapy. It should be emphasized that the mere presence of multiple varieties of organisms from cultures of any body fluid, such as from the nose and throat, does not indicate a complicated infection; in fact, when such are found, one is inclined to believe that primary infection does not exist but, more likely, that the culture was improperly secured. On the other hand, there are certain clear-cut contraindications to the use of two or more agents: allergic manifestations will be doubly apt to occur, organisms resistant to not one but two or more agents will develop and there is a great chance that there will eventually develop an infection produced by organisms completely resistant to all available drugs. Although events in each of these categories are quite uncommon, one compounds the chances for their occurrence with multiple therapies; and once a serious complication develops, there exists a distasteful and lasting impression of helplessness.

One more philosophy deserves discussion. In what manner and by what route should these very valuable agents be administered to the human? Infections in your patients are more available to direct visualization and more susceptible to local therapies than those which the physician in other fields must treat. It seems sensible then to treat locally and not systemically. As has been suggested before, antimicrobial agents are effective only as they are in direct contact with the infecting organisms which, *a priori*, are actively reproducing at the site of the infection. Generally speaking, organisms which are the most invasive and which, therefore, are actively reproducing are in greatest concentration at the base of the infected area and in the periphery of the inflammation as it extends. They are not present in significant numbers on the exposed surfaces of an ulcer or inflammation, or in the middle of collections of pus. With few exceptions, the most efficient way for an agent to reach the site of inflammation, where it can be expected to do the most good, is via the blood stream. This applies particularly to infected lesions characterized by crevices, pockets, and encrusted surfaces. I seriously question, for example, that good therapy for acute tonsillitis or acute or chronic otitis media can be as effectively treated with drops, solutions, lozenges, pastes, powders and troches, regardless of the claims made for them, as they could be with parenterally administered drugs. Perhaps when the infection is particularly avascular and blood-borne drug reaches the area in poor concentration then local therapy becomes best, but these instances are not commonly observed.

As you are well aware, I am in no position, by training or experience, to detail specific methods of treatment for most infections encountered in the ear, nose and throat and, therefore, shall not attempt to do so; but I should like to comment briefly upon three diseases which you see not infrequently and with which internists also are quite concerned, namely, septic sore throat, meningitis from sinus or otogenous foci, and tuberculosis.

Streptococcic sore throat can be treated equally well with penicillin, terramycin, aureomycin and, presumably, magna-

mycin and erythromycin. Dosage of any one of these is well described, and it makes no real difference about routes of administration. Duration of therapy, however, is a factor to be seriously considered. If the infection is treated for a minimum of five days, nonpurulent complications are rare. If less than five days of good therapy are given there is a significant increase in the incidence of both rheumatic fever and glomerulonephritis. These occur because streptolysins, which are directly related to their occurrence, are not subdued by specific antistreptococcal therapy. For safe eradication of the organisms by a combination of drug and host activities, penicillin or one of the other drugs must be given for at least five days. In practice antimicrobial therapy is too frequently stopped after 48 or 72 hours because the local inflammation has subsided and this is improper. Fortunately the insoluble forms of penicillin aid the physician by making five-day treatments easy — one dose of some types of procaine penicillin provides adequate blood levels for 48 or more hours.

Meningococcic and pneumococcic meningitis can be best treated with massive parenteral therapy with penicillin. With the use of 1,000,000 units in Q2H, continued for from 10 to 14 days, the mortality rate of meningitis from otogenic foci can be reduced to less than 25 per cent — a significant improvement over the rates generally published. The advantage of using massive parenteral therapy is to provide constant and high concentrations of the drug in spinal fluid — its diffusion from an intrathecally administered dose may be less than ideal because of fibrin blocks. As was pointed out before, single drug therapy, so long as it provides adequate concentrations at the site of infection, is as good as combined therapy.

Tuberculosis, to provide the exception, must be treated always with combined drugs — either streptomycin and para-aminosalicylic acid or streptomycin and isoniazid. One drug does not potentiate the antituberculous effects of the other, but the combination prevents the emergence of acid-fast bacilli resistant to either drug should it be used singly. The combination also apparently diminishes the unique toxicity of streptomycin.

Chemotherapy of tuberculosis must be continued for long periods, at least for six months in some and for two years in most. This is necessary primarily because tubercle bacilli, being sluggish in their growth characteristics, must be exposed to their antagonists for long periods before good control of the infection can be expected. It has been shown many times and by many observers that for these long periods dihydrostreptomycin is far more dangerous to the auditory branch of the VIIIth cranial nerve than is the parent substance. There is consequently *no* place for dihydrostreptomycin in the therapy of any kind of tuberculosis, pulmonary or ear, nose and throat.

To summarize: There are now available many agents capable of aiding the host to defend himself against invading microorganisms. Practically speaking, human infections caused by the vast majority of bacteria can be controlled with one or more drugs administered wisely. The only major exception to this general statement is the infection caused by either the proteus or pseudomonas group of Gram-negative bacteria. As primary infections, however, neither is common, and, therefore, the exception is of minor importance. On the other hand, because these organisms are resistant to commonly used antibacterial substances, their importance as secondary contaminants in difficult infections is becoming more important.

Except for stilbamidine's activity against blastomycosis, infections caused by fungi and other forms of higher microbial life are not yet treatable by specific drug therapy. Neither can infections caused by viruses be treated specifically. Effective antimicrobial therapy for these then remains for the future. As an aside, I should like to emphasize that the use of antibacterial agents for the common cold and other virus infections may soothe the physician's conscience, but their use certainly does not alter the course of the infection nor, as a matter of fact, prevent secondary infections due to bacteria.

Perhaps the practical problem facing the physician who must treat a specific bacterial infection is the choice of drug or drugs. Actually, this should be no problem, because for every one of them there is a choice of drugs, each effective and no one so superior to another that the choice can become limited. Indeed, if one drug fails to perform as expected, the answer to the failure is not the drug itself but rather to other very clear and predictable factors. Either the host is doing a poor job in defending himself; the drug is being administered wrong and is not being brought to the site of the infection; the pathology under consideration is not capable of being reversed with a drug alone — other mechanistic treatments being necessary. If one drug fails, it is highly unlikely that a second, or third or a variety of combinations will do a better job for the host and his physician. When the drug fails, the physician had best consider what is wrong in the host, not what is wrong with the drug.

OSTEOMA OF THE EXTERNAL WALL OF THE FRONTAL BONE. REMOVAL BY ENDONASAL PLASTIC APPROACH.

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Osteoma of the outer wall of the frontal bone, particularly in the region of the glabella, is rare. Those which have come to my attention have been removed by incising the skin over the tumor and excising it with the aid of a chisel and hammer under direct vision. Since an ugly scar sometimes results from this direct attack, I am reporting a new approach which eliminates that danger.

The osteoma is readily demonstrated on palpation by its configuration, consistency and location. X-ray of the frontal bone in both lateral and anteroposterior views shows a well-defined dense shadow of the tumor.

Report of a case with drawing and X-rays:

Jan. 28, 1952: Miss J. T., age 20, felt a small "pimple-like" mass over the forehead between the eyebrows four years ago. She thought nothing about its importance until one year ago when a deformity in that region was called to her attention. In the past six months the swelling had become larger, accompanied by dull, intermittent headaches between the eyes. The area was not tender to palpation. No eye symptoms such as diplopia or blurring of vision were present. There was no nasal obstruction or purulent discharge.

Past History: Measles, mumps, pneumonia. *Operation:* Tonsillectomy and adenoidectomy.

Physical Examination: Blood pressure: 120/70. Head: Hard mass, not movable or tender, was palpated over the frontal bone between the eyebrows, the size of a kidney bean. Heart and lungs were negative. Abdomen showed no masses.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Nov. 14, 1952.

Laboratory Studies: Urine: Yellow, cloudy in color. Specific gravity: 1.020. Albumin: Very slight trace. White blood cells: 5-7000. Epithelium cells: Numerous. Sugar: Negative. *Blood:* Hgb.: 11 per cent. R. B. C.: 441. W. B. C.: 5,450. Polys: 59 per cent. Lymph: 35. Monocytes: 6. Aniso: Slight. Platelets: Normal.

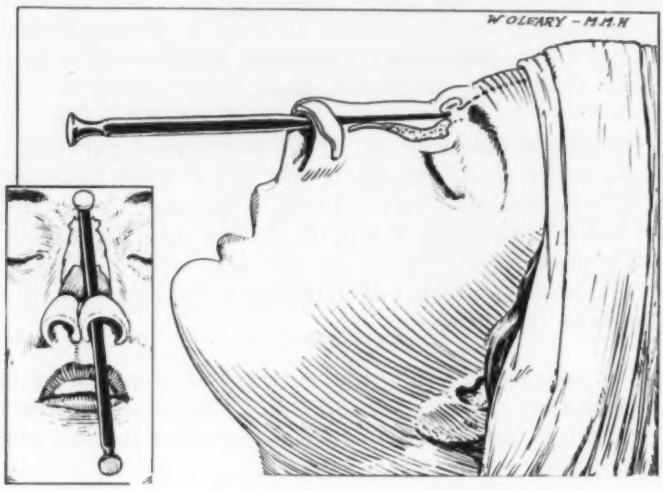

Fig. 1. Front view and side view of guarded chisel in position for removal of bony tumor.

The operation was performed on Feb. 1, 1952, using the endonasal plastic approach.

The patient was given nembutal gr. III, one and one-half hours preoperatively. The vestibule of the nose and the face was scrubbed with Septisol and the face was draped. The nose and frontal area involving the osteoma was anesthetized with 1.5 per cent monocaine in 1:30,000 adrenalin chloride solution.

An incision was made between the upper and lower cartilaginous areas on the right and left sides. The skin and subcutaneous tissue over the nose and glabella parts was elevated with double-edged Joseph knives and scissors. A No. 15 Bard-Parker knife was introduced through the wound, and the fibrous attachment around the pedicle of the osteoma was denuded. A guarded osteotome was placed beneath the tumor, as close to the frontal bone as possible, and the assistant tapped the chisel gently until the tumor was completely detached from its base. The severed tumor could now be easily grasped with a hemostat and was removed through the endonasal incision. The nose was lightly packed with sterile vaseline gauze. Externally the nose and the lower frontal bone were immobilized with adhesive tape and stent wax compound.

Fig. 2.

Fig. 3.

Condition at the end of three months shows no deformity, edema or visible scar.

Pathological Report: Gross examination: Piece of calcified, pinkish-white tissue measuring 1 x 1 x 0.5 cm. Microscopic examination: well-formed, normal looking bone. Trabecular stroma in between consists of areolar fibrous tissue. There is no evidence of infection or malignancy.

Diagnosis: Osteoma, cancellous tissue (frontal bone).

Summary and Conclusions: Osteoma of the outer wall of the frontal bone can be easily removed by the endonasal plastic approach. The advantages are: *1.* that there is no scar on the forehead following the operation, and *2.* in the hands of a nasoplastic surgeon, the approach to the tumor is very simple and direct.

475 Commonwealth Avenue.

ADDENDUM TO AN OSLER ITEM.

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Dr. Mosher's recent contribution in THE LARYNGOSCOPE, entitled "An Osler Item," could not fail to provoke interest in the younger physicians to whom Osler is a legendary figure of massive stature.

Last May while in Toronto, the writer had the opportunity of reading Dr. Mosher's manuscript before it was sent in for publication. This occasion evoked memories of a visit to the Osler Library of McGill University some years ago during a meeting of our special societies in Montreal. There, Dr. Berry, who was with me, and I found ourselves the only visitors, and the curator, Dr. W. W. Francis, quite disengaged at the time. He was most genial and helpful, not only in describing the library and its priceless volumes but also in telling us many most intriguing bits of information regarding Dr. Osler. As Dr. Francis was a nephew of his, and, as a young boy, had been brought up in his household, much of this was of an intimate nature, revealing a side of the man quite unknown, I believe, to many people.

I recalled that upon my return to the hotel that day I had made notes of what Dr. Francis had told us, which later I had amplified by certain abstracts of some of Dr. Osler's addresses. I offered this material to Dr. Mosher that he might incorporate it in his article, but with characteristic generosity he insisted that I put it in shape for publication myself; and, I fear, exercised some pressure upon Dr. Walsh to accept it.

Incidentally, sometime later, in conversation with Dr. Ireland, I was informed that Dr. Osler originally came from Toronto. For some unexplained reason nothing regarding

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Jan. 21, 1953.

Toronto had been mentioned in Montreal. Could there be geographical rivalries among our Canadian friends? Actually he was born in Bond Head in 1849 — but went to college in Toronto, after which he graduated in medicine from McGill in 1872.

It was somewhat astounding to me to learn that he was made Professor of Anatomy and Physiology at the young age of 25. From the start, private practice had little or no appeal for him, and almost always he would be found in the medical school, the hospital, or the postmortem room. On one occasion he received $500.00 for work in a smallpox hospital. This he spent on microscopes, instituting the first class in histology at McGill. He conducted this in a coatroom in the school which was available only on Saturday afternoons.

It is generally known that he personally wrote up all his autopsy findings, and that these, the results of over 1,000 postmortem examinations, furnished the basis for his valuable clinical observations. What was new, at least to me, was that each night at 10 o'clock he was accustomed to take all specimens to his old teacher, Dr. Palmer Howard, for discussion with him.

His personal life, according to Dr. Francis, was very simple but extremely well-organized. It is said that he never wasted a moment. While he was a great reader and soon began to collect fine books, he had little use for newspapers or popular magazines. He read his medical journals on the streetcars while going to and from the hospital. At meal times he read poetry aloud to the family, the book resting on a stand on the table. While dressing he made it a practice to read nonmedical literature, but while taking his bath he insisted upon being read to. This, by the way, was one of the duties of the young nephew, Francis.

Never interested in accumulating wealth, he was in such limited circumstances that when he made the move to Philadelphia he had to borrow $1,000.00; but while poor in finan-

cial resources, he was already wealthy in his knowledge of medicine and his philosphy of life, which his stern mental disciplines had developed in him.

His forte was clinical teaching and he developed bedside instruction as we know it today. His lectures never were of the formal type. It was said that he did not conform to the Philadelphia traditions of the day. He was not especially happy there and welcomed the opportunity to go to the young Johns Hopkins Hospital, which he felt was free of tradition and quite unfettered. He was there in the hospital for five years before the medical school was established. This gave him considerable time which he utilized for writing.

In 1905, at the age of 56, feeling he was wearing himself out, he left Baltimore for Oxford, where he was made Regius Professor of Medicine. He said that all progress was made, or at least started, before 40 and that a man should retire from active work at 60. The lack of clinical teaching at Oxford was a source of regret and disappointment to him, and one wonders if the last 14 years of his life would not have been happier had he been able to continue in his favorite vocation.

It was inevitable that, as time went on, Osler should be known best by his writings; for he left a priceless heritage, not alone to medicine but to mankind generally. While "Aequanimitas" was addressed to the graduating class at the University of Pennsylvania, this, as does the Yale essay, "A Way of Life," carries a message to all, layman as well as physician; and "The Master-Word in Medicine" embodies a lesson which is not limited to the profession: "And the Master-Word is Work, a little one, as I have said, but fraught with momentous consequences if you can but write it on the tables of your heart and bind it upon your forehead."

To medicine specifically he bequeathed an idealism which should never be forsaken. "The practice of medicine is an art, not a trade; a calling, not a business; a calling in which your heart will be exercised equally with your head," and, "I have these personal ideals: one, to do a day's work well

and not bother about it tomorrow. . . . The second ideal has been to act the Golden Rule, as far as in me lay, toward my professional brethren, and toward the patients committed to my care. And the third has been to cultivate such a measure of equanimity as would enable me to bear success with humility, the affection of my friends without pride, and to be ready when the day of sorrow and grief comes to meet it with the courage befitting a man."

Yet there seems to have been another side to him — not so generally recognized. This master-teacher, this writer with an unsurpassed gift for language, this philosopher of medicine was not always the serious, austere personage that we might imagine.

One of his old students, Dr. Joseph H. Pratt, told me of an incident that occurred many years ago at a meeting of one of the national medical societies in Boston. Dr. Osler arranged for a private dinner at one of the clubs for a few select friends. These included names that loom large in medical circles, and it was a privilege for Pratt, then a very young man, to be included. Desirous of providing a typical Boston repast, Osler had selected scrod for the entree. One of the distinguished guests had never heard of it and, being impressed by its flavor, asked just what scrod might be. Whereupon Osler launched on a long and fanciful discourse, all with most serious mien, on the habits and cultivation of scrod, implying that the common codfish went upstream to spawn where they were trapped, and, after being treated surgically, were kept in pools and allowed to grow; and that, finally, the scrod was a "caponized" cod.

To Dr. Francis we were indebted for the interesting information that Osler frequently indulged in anonymous authorship under the name of Davis, writing the weirdest type of fiction as an amusement. Perhaps the outstanding example of this was his story of the "Dismal Swamp."

Intrigued by Tom Moore's poem, "The Lake of the Dismal Swamp," Osler, accompanied by a close friend, paid a visit to the lake at the time of construction of the Albemarle Canal.

They travelled by launch through the outlet of the lake with banks eight to 10 feet high until, passing through a small lock, they reached the lake itself. The scene of utter desolation, with innumerable cypress trees with their roots rising out of the water, and merging to form trunks several feet above the level of the lake, must have made a vivid impression upon Osler. While eating lunch he proceeded to write a most fantastic story of his experiences, using the blank pages and margins of a copy of Burton's "Anatomy of Melancholy" which he had with him. In this he described how brilliant-hued moccasin snakes dropped into the boat from the limbs of trees, of how they had met a man with a "vertical eye" and of Negroes who had not heard of the war between the states and the emancipation of slaves, and who still thought George Washington was President.

This copy of Burton with Osler's story of the "Dismal Swamp" is in the Osler Library at McGill, where we had the privilege of seeing it.

To me, at first, it seemed difficult to reconcile this form of writing, so utterly fictitious and fantastic, with the scholarly philosophic addresses, the precise descriptions of his text-book, so carefully edited and re-edited, and the shrewd practical lessons drawn from his powers of observation and so frequently quoted as aphorisms; and then it became evident that this profound thinker possessed a keen sense of humor — not easily discernible to a later generation, and that it sought its outlet in that field in which he excelled and was most at home, namely, in writing. To me, at least, this realization vitalized a formerly legendary figure and put added meaning to his writings.

PLASTIC AND RECONSTRUCTIVE SURGERY.

A Review of the Literature for 1951.*†

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Gillies[1] reports the probability of success for grafting preserved ox cartilage to the nose as 95 per cent; for grafting it to the ear, 50 per cent; for restoring other facial contours with it, 80 per cent. He, therefore, recommends its use, particularly for reconstruction of the nose where results are comparable to any other grafting material. According to Peer[2,3] human rib, tibial, and iliac bone grafts, when placed in contact with unlike tissues, are all replaced in six to eight months, regardless of presence or absence of periosteum on the grafts. Many bone cells survive, but their ultimate fate cannot be determined, as they disappear in a multitude of invading fibroblasts. He[4] believes the cells in cartilage, septal bone, fascia, tendon and surface skin grafts all tend to survive, and that the tissues retain their specific structure without mass replacement of graft cells by infiltrating elements from the host tissues. For buried skin grafts, Gray *et al.*[5] and Browne[6] recommend using cutis rather than whole skin as many objectionable features of whole skin, such as inclusion cysts and keratolytic debris, can be eliminated.

SCARS AND HEALING.

By an ingenious experiment Min-Chyang Ju[7] has illustrated the physical basis of scar contracture, explaining the deformity associated with trapdoor type flaps, and explaining why scars parallel to and scars perpendicular to Langer's lines

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†Reprinted in part from Cyclopedia of Medicine, Surgery and Specialties, pp. 717-734, 1952.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, July 29, 1952.

behave differently. Elective incisions on the body surface run uniformly perpendicular to the action of the underlying muscles upon which they are dependent for their formation. Kraissel[8] states that this pattern of lines may be demonstrated by existent wrinkles, by gentle compression of the relaxed skin and by contraction of the muscles in the region concerned. These lines might be said to be created by dynamic forces and may be at some variance with Langer's lines, which are the result of a study of static forces acting on the cadaver skin.

Keloids and hypertrophic scars are not true tumors but are a peculiar type of foreign body granuloma, according to Gluckmann.[9] Control of hypertrophic scars, Mowlem[10] thinks, might be achieved theoretically by desensitization to the local foreign body keratin or more practically by reduction of activating factors such as exogenous and endogenous foreign material. Levitt[11] recommends pre- and postoperative radiotherapy for hypertrophic scars as this inhibits epidermal proliferation, thereby lessening endogenous foreign body formation; also, it reduces vascular supply and inflammatory cellular infiltration of the dermis.

Edgerton, Hanrahan and Davis[12] have given vitamin E prophylactically to prevent keloids, but it has not been effective except in relieving subjective discomfort.

On the experimental animal, administration of cortisone and ACTH have been found to inhibit healing of incised wounds.[13,14] In man, there may be some delay in primary wound healing, but usually this is not the case.[15,16]

EYELIDS.

An operative procedure for correction of a large central coloboma of the upper lid is described by Callahan.[17] It consists of reducing by half the horizontal dimension of the coloboma, into which defect is free-grafted a section of the entire thickness of the other upper lid. Manchester[18] repairs full thickness losses of the lower lid by raising a conjunctival flap from the fornix and lower bulbar conjunctiva and then swinging down over it a double pedicle flap from the upper lid.

Residual eversion of the inferior lacrimal punctum can be corrected by a procedure outlined by Lee[19] in which the lacrimal portion of the upper and lower lid margins are fused. Senile entropion of the lower lid can be helped by the method of Fox[20] in which a tarsal triangle with apex up is excised and sutured so that the base of the tarsus is shortened. Excess skin is removed also.

Herniation of intraorbital fat is not uncommon. Under the upper lid there are two separate and independent pockets of fat, and under the lower eyelid there are three similar ones. Castanares[21] emphasizes the importance of removal of the fat from all pockets involved in order to obtain a satisfactory result. The external skin approach is used rather than through the conjunctiva.

Blepharochalosis is a rather uncommon condition limited to upper lids and consisting of thin, relaxed, extremely loose excess skin. This redundant tissue is excised and sutured to upper border of the tarsus as suggested by Fitche and O'Connor.[22]

CLEFT PALATES.

A new procedure to close and elongate the partial cleft palate is designed by Cuthbert,[23] who raised three mucoperiosteal flaps instead of two and by this means achieved greater posterior displacement of palate and less chance of fistula formation. Moran[24] has used the pharyngeal flap operation effectively as a speech aid in a group of 35 secondary palate cases. Excellent speech was achieved in eight, good speech in 15, and fair or improved speech in nine. Whether the flap is based above or below depends on the amount of adenoid tissue present and the distance from the pharyngeal wall to the palate. Similar favorable results following this procedure have been confirmed by Dunn.[25] A combination push-back operation and pharyngeal flap operation has been reported by Conway[26] in 38 cases which were complicated by severe scarring from previous unsuccessful surgery.

When surgical attempts at closure of a palate have failed, Harkins[27] requests a redivision of the soft palate before proceeding with rehabilitation by means of prosthodontia.

Early intervention by the orthodontist can guide the developing dentition into areas of good function with the result that major deformities of the past are not permitted to occur. Conroy[28] undertakes orthodontic therapy whenever growth and denture positions indicate that an improvement can be expected and the cooperation of the child can be secured.

Beatty[29] still advocates using the old Hudson-Macuen operation as taught by Brophy, by which the posterior tonsillar pillars are sutured together as a means of repair after tonsillectomy.

An excellent anatomical discourse on the nerve supply of the soft palate is given by Broomhead,[30] and it is shown that damage to the nerve to the tensor palati may be inflicted during surgery, thus paralyzing this muscle. Repair of a congenital atresia of the oropharynx is reported by Longacre.[31]

EARS.

A complete list and description of all conceivable malformations of the auricle and external auditory meatus is given by Altmann.[32] The conventional repair of prominent ears by Jayes and Dale[33] after Luckett is to be contrasted with a quick, easy repair described by Gonzalez-Ulloa.[34] The latter is done by dividing the cartilage through a small anterior skin incision and by the use of transfixion sutures through the cartilage. No dressing is necessary. For scoop-like ears Ragnell[35] splits the cartilage zigzag-wise into serrations 3 to 4 mm. wide and then resutures them together as a lattice-work in the desired shape.

Four cases with congenital atresia of the external auditory meatus have been operated by Davis,[36] who creates a direct bony canal to the antrum. At a second stage this cavity is lined with a skin graft applied closely to the external semicircular canal, thus permitting improved hearing.

NOSE.

Brown and McDowell[37] give a very complete work on plastic surgery of the nose including surgical reduction in the

size of the nose, building up and straightening the nose, correction of nasal deformities associated with cleft lips, and repairs of various defects with grafts and flaps. In routine rhinoplasty it is recommended that the lower lateral cartilages be trimmed by the eversion technique, that is, through the original intercartilaginous incisions rather than making accessory rim incisions. This procedure is a little more difficult but it leaves a nicer nostril border without scars. The cartilage is swung out of the nostril on a hinge of skin along the nostril rim. The upper border of the cartilage is thereby available for trimming. For alleviating the typical porcine appearance in double cleft lip deformities, a partial incision of the nostril base is done. For early treatment of frontal "squashed" nasal fractures which cannot be held by an aluminum splint alone, through-and-through 24 stainless steel wires are placed behind the nose, tied over lead places laterally to hold the lateral walls inward and the dorsum forward. The use of flaps for nasal reconstruction is illustrated, but it is required less now because small partial repairs can be done using composite grafts from the ears.

A simplified procedure for elective rhinoplasty is outlined by Bames.[38] Fomon[39] obtains better results by altering the shape of the lower lateral cartilages than by sacrificing their substance when the bulk of the cartilage is in normal limits. In cases with very large nasal tips, the Rethi incision across the columella can be used for better exposure and without much additional scarring according to May.[40] Problems of the septum are handled by Becher[41] by vertical cutting and removal of narrow sections of cartilage. Fomon[42] clears the airway by realigning, unbuckling, and removing part or all of the deflected portion of the septum. Cohen[43] approaches postrhinoplastic adhesions by shifting local tissue flaps and, if not feasible, then by grafting skin.

A simple two-stage operation involving a local caterpillar flap brought down by a V-Y procedure is used by Cronin[44] to reconstruct a nasal tip. Subtotal rhinoplasty repairs by composite grafts are limited by the size of the defects so that contiguous skin flaps from cheeks and forehead may be neces-

sary; however, as Lewin[45] points out, additional scarring limits the usefulness of these flaps. A median frontal flap is brought down by Cardoso[46] to correct the retraction of the lower half of the nose in cases of leishmaniasis.

MANDIBLE.

A serial study of mandibular growth after removal of the condyle was done on the Macaca Rhesus monkey by Sarnat and Engel.[47] This revealed a serious interference in growth of the mandible, particularly in the vertical growth of the ramus. The consequences of injury to the condylar growth center may be even more extensive in the human than in the experimental animal; therefore, operative interference in this area in children should be undertaken only after consideration of its possible effects on mandibular growth. If ankylosis of the jaw occurs before cessation of growth at condylar growth center (16 to 18 years) there will be loss of function and hence underdevelopment of the mandible and of the face; therefore, Pickrell and associates[48] urge correction by a modified Risdon type ostectomy with interpolation of a muscle flap. Longacre and Gilby[49] use autogenous cartilage graft in the ramisection gap for true ankylosis of the temporomandibular joint.

Stuart Gordon[50] has a new technique for stabilizing a recurrent dislocating temporomandibular joint by an external lateral ligament of fascialata threaded vertically through the zygomatic arch and horizontally through the condyle twice and tied on itself. The interarticular cartilage is removed from the joint first.

Selected cases of prognatism are thought by Gonzalez-Ulloa[51] to be better treated by removal of the condyle bilaterally. The operative approach and procedure are described.

The successful use of vitallum prostheses and implants in jaw reconstruction is attested by Conley[52] and Kleitsch.[53]

FACIAL INJURIES AND RECONSTRUCTIVE SURGERY OF THE FACE.

Brown, Fryer and McDowell[54] use internal wire-pin stabilization for middle third facial fractures. A Kirschner wire

is inserted through the face for a separated maxilla on one side, for fractures through the palate or for loosened zygomas. Immediate stabilization is achieved by cantilever support. This may be used in combination with direct wiring of zygoma to the frontal bone, through-and-through wiring fixation of the nose, interdental wiring, and open elevation of orbital borders.

According to Murphy *et al.*[55] external fixation and extension is the most satisfactory method of handling maxillary fractures in regard to comfort and absolute fixation of fragments. Tanzer[56] employs a head cap and balanced elastic support as a means of fixation of malar and some maxillary fractures. Treatment of malunion of the zygomaticomaxillary compound can be managed by osteotomy and realignment of the fragments. In cases of not too old fibrous malunion of the maxilla, Dingman[57] describes a new procedure referred to as the impression tray method of refracture.

In severe deformities supportive grafts may be required in late repairs. In a few cases, Aldunate[58] has squirted acrylic paste into facial and skull depressions; but a continued search is being made for cartilage and bone graft substitutes for filling in depressions in contour. Polyethylene as shown by Rubin[59] causes minimal reaction. A new light weight, wettable, resilient polyvinyl sponge has been reported by Grindlay and Waugh[60] as being implanted subcutaneously without evidence of inflammatory reaction. This would presumably present possibilities for plastic repair.

Fibrous dysplasia of the maxilla presents a plastic surgery problem and is best treated by surgical removal and X-ray therapy.[61] VonDeilen[62] presents a case of bilateral maxillary amputation for carcinoma with immediate skin grafting of the defect and contour restoration by early insertion of an appliance.

Plastic surgery repairs for traumatic soft tissue facial disfigurements are managed by Soderberg[63] by replacement by

local rotation or advancement flaps, by pedicle flaps, and by free graft transplants. The primary purpose should be restoration of function with cosmesis as a secondary factor.

A face-lifting procedure characterized by rotation flaps has been designed by Fomon, Goldman, Neivert and Schattner.[64] They tighten the fascia by tucks taken at strategic points and the cheek is supported by two sutures introduced at the limit of skin undermining and looped over the zygoma. A triangle of skin from the scalp above the ear is removed in addition to the usual excisions.

An excellent method for repairing cranial defects is with fresh autogenous iliac bone, according to McClintock and Dingmann.[65] It is physiologically sound and readily available; there is adequate quantity; contouring is easy; and one can still visualize air or arteriographic studies through it. Infections and foreign body reactions are less likely.

Skin grafting the scalp may be easier by making a free pattern of the defect. By allowing the pattern to flatten out, one is given an ideas as to the amount of skin necessary for coverage.[66]

FACIAL PARALYSIS.

For facial paralysis, Brunner[67] combines the masseter plastic operation and a procedure for support of the angle of the mouth. Both operations can be performed intraorally. In the latter, kangaroo tendon or catgut is used as a sling for anchorage of the soft tissue about the corner of the mouth to the frontal process of the maxilla.

SALIVARY GLAND.

For all recurrent and for most primary mixed tumors of the parotid gland, McCune[68] does a total parotidectimy. He reports nerve paralysis in five out of 27 cases operated. Three of these paralyses were in patients who had had a recurrence. On the other hand, Kirklin and associates[69] and Hendrich, Ward and Lacy[70] believe that primary mixed tumors require only adequate local excision and not subtotal or

total parotidectomy as advocated by Bailey for all mixed tumors. This is because 60 to 70 per cent are not going to have a recurrence and, therefore, are spared the risk of damage to the VIIth nerve. For recurrences, wide local excision or total paroidectomy is warranted, as the risk of another recurrence is definitely higher (40 to 50 per cent).

HEAD AND NECK SURGERY.

Ward and Hendrick's[71] text on tumors of the head and neck is a most valuable book to the profession, as it embodies the authors' wide experience with malignancies in this region. The treatment of oral cancer by a combination of radiotherapy and surgery has given a 33 per cent five-year survival for carcinoma of the tongue and a 58 per cent five-year survival for carcinoma of the lip in a series of cases by Woolsey, Ash, Welsh and Mustard.[72] Carcinoma of the tongue is treated by Kremen[73] and Barclay *et al.*[74] by primary combined en bloc resection of tongue, floor of the mouth and cervical lymphatics, sparing the mandible if possible. Prophylactic neck dissections were done on most patients with carcinoma of the oral cavity except the lip. For resection of malignant lesions in and about the mouth Sugarbaker and Wiley[75] found simultaneous tourniquet or temporary ligation of both external carotid arteries helpful in decreasing bleeding and in facilitating exposure.

A definite increase in intracranial pressure occurs after radical neck dissections and is more pronounced and persistent after removal of the second internal jugular. Jones[76] warns against repeated lumbar punctures and points out the danger of compression of the opposite internal jugular by the dressing. It is recommended that the patient be kept in a sitting position and if necessary hypertrophic dextrose solutions be given intravenously during the critical period.

Blood volume determinations and blood loss measurements of Royster and associates[77] on patients having radical neck dissections revealed a blood volume deficit in the majority. It was thought that preoperative blood transfusions in deficit patients would be helpful in preventing shock.

Three cases of congenital defect derived from the first branchial cleft are presented by Byars and Anderson.[78] This anomaly is found high in the neck, above the level of the hyoid, below the lower border of the mandible and probably opening into or in close proximity to the external auditory canal. Diagnosis is made if there is a cystic swelling or draining fistula high in the neck with accompanying ear discharge without middle ear infection or a ruptured drum. Removal requires two incisions, one in the neck and one anterior to the ear, with careful dissection of the tract to avoid the facial nerve.

The thyroglossal duct is resected from the hyoid to the foramen cecum by Fallon[79] by using a skewer (a heavy needle) placed between these two locations and over it passing a corer (a tubular knife).

Unless biopsy shows the tumor to be malignant, Lahey and Warren[80] do not recommend removal of carotid body tumors with ligation of the internal, external or common carotid arteries would be required. This is particularly sound advice in view of the fact that very few have been found to be malignant. Heparin and dicumerol should be used when ligation of the internal or common carotid is done.[81]

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BOOK REVIEWS.

Ménière's Disease. By Henry L. Williams, M.D., Head, Section of Otolaryngology and Rhinology, Mayo Clinic; Professor of Otolaryngology and Rhinology, Mayo Foundation; Graduate School of University of Minnesota, Rochester, Minn. 350 pages with index and 15 figures. 1952. Springfield, Ill.: Charles C. Thomas, Publishers.

This is a well executed volume; the printing is clear, the paper is good and the binding pleasant.

This most complete dissertation on Ménière's Disease is what this reviewer expected from the author. Each phase of the subject is dealt with fully; at the end of each chapter there is a complete bibliography, and it is refreshing that the references have been read in the original and accurately quoted. The author is justly critical of misquotations by others.

Chapter Six, entitled Fundamental Identity of Allergy and Autonomic Dysfunction, is excellent and should be required reading by all who practice otolaryngologic allergy.

The chapter on the medical management of Ménière's Disease will prove helpful to the otologist confronted by these baffling and difficult conditions. The treatment of the surgical management is clear and concise.

We congratulate the author on this excellent monograph. It should prove invaluable to students and warrants a place in the library of every practicing otologist. T. E. W.

Auditory Training for the Deaf. Mary Wood Whitehurst and Edna K. Monsees. Volta Bureau, Washington 37, D. C. 1952. Soft cover, 99 pp. $3.00.

This book is essentially a manual for teachers to provide a method and basic lesson material for auditory training of the deaf. The authors, both experienced in working with individuals with hearing impairment, have adapted many of the lessons for home study and practice by the pupil himself with the aid of an assistant. In general the lessons furnish material for listening experiences in sound contrasts, sounds in words, sentences and songs, and rhythmic patterns. It is felt that these experiences will enable the child with a residuum of hearing to recognize sounds and thus to improve his understanding of speech. Combined with lipreading, the training in recognition of sounds should enhance the information carried to the brain. The manual is in the tradition of Urbantschitsch's "Uber Hörübungen bei Taubstummheit," which appeared in the 1890's in Vienna, and Goldstein's "The Acoustic Method" (1939), although not so elaborate in treatment.

On the other hand, the material of Whitehurst and Monsees implies the use of improved hearing aids not available to the previous writers on the subject. With improving performance of hearing aids, their proper and effective use becomes increasingly important. The manual, therefore, fills a need which should enable teachers to take advantage of modern instrumentation for auditory training. The book contains a brief but informative foreword on the principles of auditory training by Dr. William G. Hardy, Director, Hearing and Speech Center of the Johns Hopkins Medical Institutions. The professional affiliations of the authors are not given. S. R. S.

Jane Walker's Book of Art Lectures for Lipreading Practice. Volta Bureau, Washington 37, D. C. 1951. Soft cover, 61 pp. $3.12 postpaid.

This compendium of lipreading practice material combines the background as a teacher of art and of lipreading of the late Jane B. Walker. It contains a wealth of stimulating material about art for the teacher of lipreading who is always looking for subject matter which can keep her students motivated. S. R. S.

BRONCHOESOPHAGOLOGY COURSE.

The next Bronchoesophagology Course to be given by the University of Illinois College of Medicine is scheduled for the period March 16 through March 28, 1953. The course is under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Ill.

ALUMNI ASSOCIATION — NEW YORK EYE AND EAR INFIRMARY.

The Alumni Association of the New York Eye and Ear Infirmary will hold its annual scientific program starting April 20 through April 24. The scientific sessions on ophthalmology will be held Monday and Tuesday. Sessions on ear, nose and throat on Thursday and Friday at the New York Academy of Medicine in Room 440. Morning sessions will commence at 9:30, afternoon sessions at 2:00. Wednesday will be left open for clinical and surgical demonstrations at the New York Eye and Ear Infirmary.

For further information, write Dr. Hunter H. Romaine, Secretary-Treasurer, 218 Second Avenue, New York 3, N. Y.

AMERICAN ASSOCIATION FOR CLEFT PALATE REHABILITATION.

The annual meeting of the American Association for Cleft Palate Rehabilitation will be held at the Atlanta-Biltmore Hotel in Atlanta, Ga., Monday and Tuesday, April 27 and 28. The program will include special sectional meetings in the areas of medical-surgical problems, dental-prosthetic-orthodontic problems, and speech - psychology - education - social

work. Motion pictures and special exhibits relating to cleft palate rehabilitation will be shown throughout the convention. The meeting will be open to any individual with an interest in cleft palate rehabilitation. Further information about the program may be obtained from Dr. Willard T. Hunnicutt, 302 Medical Arts Building, Atlanta, Ga. Further information about membership in the association may be obtained from the Secretary, Dr. Jack Matthews, 1617 Cathedral of Learning, University of Pittsburgh, Pittsburgh 13, Pa.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

AND

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY JOINT MEETING.

Arrangements have been completed for a joint meeting of the South Carolina Society of Ophthalmology and Otolaryngology and the North Carolina Eye, Ear, Nose and Throat Society to be held at the Francis Marion Hotel, Charleston, S. Car., Sept. 14, 15 and 16, 1953. The following otolaryngologists will be on the program: Dr. Wayne Slaughter, of Chicago; Dr. John R. Lindsay, of Chicago; Dr. Theodore Walsh, of St. Louis.

A panel on vertigo will be discussed from the opththalmological, otolaryngological, neurological and internal medical standpoints.

Dr. Charles Kunkle, of Duke University, will present neurology, and Dr. Vince Mosely, of the Medical College of South Carolina, will be the guest internist.

The following three will cover the ophthalmological subjects: Dr. Derrick Vail, of Chicago; Dr. David G. Cogan, of Boston; Dr. R. Townley Paton, of New York.

For further information, please communicate with Dr. George T. Ferguson, Secretary of the North Carolina E. and T. Society, care of McPherson Hospital, Durham, N. Car., or Dr. Roderick Macdonald, Secretary of the South Carolina Society of Ophthalmology and Otolaryngology, 330 East Main Street, Rock Hill, S. Car.

WORLD MEDICAL ASSOCIATION CONFERENCE.

Dr. Julius Lempert, Director of the Lempert Institute of Otology, New York, will be guest-participant in panel discussions at the First Western Hemisphere Conference of the World Medical Association at Richmond, Va., on April 24, 1953.

The sessions will provide opportunity for a discussion of current medical problems between representatives of the national medical societies of Latin America and United States specialists and practitioners. Dr. Louis H. Bauer, President of the American Medical Association and Secretary-General of the World Medical Association, will moderate at a general session at which panel reports will be made.

Dr. Lempert will contribute a chapter on major advances in the field of otolaryngology to a volume commemorating the conference. He is a diplomate of the American Board of Otolaryngology; a member of the American Academy of Ophthalmology and Otolaryngology; a Fellow of the American Laryngological, Rhinological and Otological Society, Inc., and of the American Otological Society, Inc., and a graduate of the Long Island College of Medicine, Brooklyn, N. Y.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

February, 1953.

Audicon Models 400 and 415.

Manufacturer: National Earphone Co., Inc., 20-22 Shipman St., Newark 2, N. J.

Auditone Model 11.

Manufacturer: Audio Co. of America, 5305 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67.

Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Symphonette; Beltone Mono-Pac Model M.

Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Model 700; Cleartone Regency Model.

Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Model D-1; Dahlberg Junior Model D-2; Dahlberg Model D-3; Dahlberg Model D-4.

Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Dysonic Model 1.

Manufacturer: Dynamic Hearing Aids, 149 Church St., New York 7, N. Y.

Electroear Model C.

Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.

Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.
Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico UE-Atomeer; Maico Quiet Ear Models G and H; Maico Model J.

Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis 1, Minn.

Mears (Crystal and Magnetic) Aurophone Model 200; Mears Aurophone Model 98.

Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 303; Micronic Model "Mercury"; Micronic Star Model.

Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone T5 Audiomatic; Microtone Classic Model T9; Microtone Model T10; Microtone Model T612; Microtone Model 45.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

National Cub Model C; National Cub Model D (Duplex); National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Normatone Model C.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40 St., Minneapolis, Minn.

Distributor: Normatone Hearing Aid Co.

Otarion Model E-4; Otarion Models F-1, F-2 and F-3; Otarion Model G-2; Otarion Model G-3.

Manufacturer: Otarion Hearing Aids, 4757 N. Ravenwood, Chicago 40, Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny-Mite); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55; Radioear Model 62 Starlet; Model 72.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Distributor: Radioear Corp.

Rochester Model R-1; Rochester Model R-2.

Manufacturer: Rochester Acoustical Laboratories, Inc., 117 Fourth St., S.W., Rochester, Minn.

Silvertone Model J-92; Silvertone Model P-15.

Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.

Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 500; Telex Model 952; Telex Model 1700.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Tonamic Model 50.

Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster Model Royal; Model Cameo.

Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill

Unex Midget Model 95; Unex Midget Model 110; Unex Models 200 and 230.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Models J and J-2.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Models 65 and 66.
Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Ambco Hearing Amplifier (Table Model).
Manufacturer: A. M. Brooks Co., 64 S. Bonnie Brae St., Los Angeles 5, Calif.

Aurex (Semi-Portable).
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago 10, Ill.

Precision Table Hearing Aid.
Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. Albert C. Furstenberg, University Hospital, Ann Arbor, Mich.
Vice-President: Dr. Frederick T. Hill, Professional Bldg., Waterville, Me.
Secretary: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Ill.
Meeting: Roosevelt Hotel, New Orleans, La., May 1-2, 1953.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. H. Marshall Taylor, 111 W. Adams St., Jacksonville, Fla.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2, Pa.
Meeting: Roosevelt Hotel, New Orleans, La., Apr. 26-27, 1953.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. F. E. LeJeune, Ochsner Clinic, New Orleans, La.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Meeting: Roosevelt Hotel, New Orleans, La., April 28-29, 1953 (mornings only).

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. Carl H. McCaskey, 608 Guaranty Bldg., Indianapolis, Ind.
Vice-Chairman: Dr. Fred W. Dixon, Rose Bldg., Cleveland, Ohio.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3, Tenn.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James M. Robb, Detroit, Mich.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., Oct. 11-17, 1953.

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Roosevelt Hotel, New Orleans, La., Apr. 21-25, 1953.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Herman J. Moersch.
Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1, Md.
Meeting: Roosevelt Hotel, New Orleans, La., Apr. 28-29, 1953 (afternoons only).

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Clifton E. Benson, Bremerton, Wash.
President-Elect: Dr. Carl D. F. Jensen, Seattle, Wash.
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

THE SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. Victor Alfaro.
Vice-Chairman: Dr. Irvin Feldman.
Secretary: Dr. Frasier Williams.
Treasurer: Dr. John Louzan.
Meetings are held on the third Tuesday of October, November, March and May, 7:00 P.M.
Place: Army and Navy Club, Washington, D. C.

THE LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. W. L. Hughes, Lamar Life Bldg., Jackson, Miss.
Vice-President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Meeting: Edgewater Gulf Hotel, Edgewater, Miss., May 11, 1953.

OTOSCLEROSIS STUDY GROUP.

President: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Ill.
Secretary: Dr. Lawrence R. Boies, Med. Arts Bldg., Minneapolis 2, Minn.
Meeting: Palmer House, Chicago, Ill., Oct. 11-17, 1953.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis, Ind.
President-Elect: Dr. Albert D. Ruedemann, 1633 David Whitney Bldg., Detroit 26, Mich.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2, N. Y.
Meeting: Palmer House, Chicago, Ill., Oct. 16, 1953.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Justo M. Alonso, Montevideo.
Executive Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U. S. A.
Meeting: Fourth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
President: Dr. Ricardo Tapia Acuna, Mexico City.
Time and Place: Feb. 28 to Mar. 4, 1954, Mexico City.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Daniel L. Sexton, St. Louis, Mo.
President-Elect: Dr. John I. Marker, Davenport, Iowa.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.
Meeting:

THE VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. D. A. Morgan, Norfolk, Va.
Secretary-Treasurer: Dr. G. Slaughter Fitz-Hugh, Charlottesville, Va.
Spring Meeting: Hot Springs, Va., May, 1953.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Victor Goodhill.
Secretary-Treasurer: Dr. Orwyn Ellis.
Chairman of E. N. T. Section: Dr. Harold Owens.
Recording Secretary, E. N. T. Section: Dr. Donald B. Hull.
Chairman of Eye Section: Dr. Deane Hartman.
Recording Secretary, Eye Section: Dr. Robert Norene.
Time: 6:00 P.M., fourth Monday of each month from September to May, inclusive.

THIRD LATIN AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY AND BRONCHOESOPHAGOLOGY.

President: Dr. Franz Conde Jahn.
Vice-Presidents: Drs. Julio Garcia Alvarez, Angel Bustillos and Celis Perez.
Secretary General: Dr. Victorino Marquez Reveron.
Secretary of Assemblies: Dr. Cesar Rodriquez.
Time and Place: Caracas, Venezuela, July 31, 1954.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Norman N. Smith, 291 Whitney Ave., New Haven 11, Conn.
Secretary: Dr. Joseph G. Gilbert, 111 E. 61st St., New York 21, N. Y.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. William Banks Anderson, Durham, N. Car.
Secretary and Trasurer: Dr. Geo. B. Ferguson, Durham, N. Car.
Meeting: Charleston, S. Car., Sept. 13-16, 1953.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Clay W. Evatt, Charleston, S. Car.
Vice-President: Dr. David S. Asbill, Columbia, S. Car.
Secretary-Treasurer: Dr. Roderick Macdonald, Rock Hill, S. Car.
Meeting, Joint: Charleston, S. Car., Sept. 13-16, 1953.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Dr. C. Allen Dickey, 450 Sutter St., San Francisco, Calif.
Secretary-Treasurer: Dr. Howard P. House, 1136 W. Sixth St., Los Angeles 17, Calif.
Meeting: Los Angeles, Calif., 1953.

THE RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Chairman: Dr. Isaac H. Jones, 635 S. Westlake, Los Angeles, Calif.
Treasurer: Dr. Pierre Violé, 1930 Wilshire Blvd., Los Angeles, Calif.
Program Chairmen:
Otolaryngology: Dr. Leland G. Hunnicutt, 98 N. Madison Ave., Pasadena, Calif.
Ophthalmology: Dr. Harold F. Whalman, 727 W. 7th St., Los Angeles, Calif.
Mid-Winter Clinical Convention annually the last two weeks in January at Los Angeles, Calif.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Chas. C. Grace, 145 King St., St. Augustine, Fla.
President-Elect: Dr. Jos. W. Taylor, 706 Franklin St., Tampa, Fla.
Secretary-Treasurer: Dr. Carl S. McLemore, 1217 Kuhl Ave., Orlando, Fla.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President: Dr. Harry P. Schenck.
Vice-President: Dr. William J. Hitschler.
Treasurer: Dr. Chevalier L. Jackson.
Secretary: Dr. John J. O'Keefe.
Historian: Dr. Herman B. Cohen.
Executive Committee: Dr. M. Valentine Miller, Dr. Charles E. Towson, Dr. Thomas F. Furlong, Dr. Benjamin H. Shuster, ex-officio.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Vice-Chairman: Dr. K. W. Cosgrove, 111 E. Capitol Ave., Little Rock, Ark.
Secretary: Dr. F. A. Holden, Medical Arts Bldg., Baltimore, Md.
Meeting:

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Melvin W. McGehee, 425 Eleventh St., Huntington 1, W. Va.
President-Elect: Dr. James K. Stewart, Wheeling, W. Va.
Secretary-Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.
Meeting: Hot Springs, Va., May 2-3, 1953.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. C. Otrich, Belleville, Ill.
President-Elect: Dr. Phil R. McGrath, Peoria, Ill.
Secretary-Treasurer: Dr. Alfred G. Schultz, Jacksonville, Ill.

CANADIAN OTOLARYNGOLOGICAL SOCIETY SOCIETE CANADIENNE D'OTOLARYNGOLOGIE

President: Dr. D. E. S. Wishart, 170 St. George St., Toronto, Ontario.
Secretary: Dr. W. Ross Wright, 361 Regent St., Fredericton, N. B.
Place: Minaki Lodge, Minaki, Ontario.
Time: June 14-18, 1953.

SOCIEDAD DE OTO-RINO-LARINGOLOGIA, COLEGIO MEDICO DE EL SALVADOR, SAN SALVADOR, C. A.

President: Dr. Victor M. Noubleau.
Secretary: Dr. Héctor R. Silva.
1o. Vocal: Dr. Salvador Mixco Pinto.
2o. Vocal: Dr. Daniel Alfredo Alfaro.

MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

President: Dr. Cesar LaBoide, Mexico, D. F.
Vice-President: Dr. M. Gonzalez Ulloa, Mexico, D. F.
Secretary: Dr. Juan de Dios Peza, Mexico, D. F.

FEDERACION ARGENTINA, DE SOCIEDADES DE OTORRINOLARINGOLOGIA.

Secretario del Exterior: Dr. Juan Manuel Tato.
Sub-Secretario del Exterior: Dr. Oreste E. Bergaglio.
Secretario del Interior: Dr. Eduardo Casterán.
Sub-Secretario del Interior: Dr.Atilio Viale del Carril.
Secretario Tesorero: Dr. Vicente Carri.
Sub-Secretario Tesorero: Dr. José D. Suberviola.

ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

Presidente: Dr. J. Abello.
Vice-Presdente: Dr. Luis Suñe Medan.
Secretario: Dr. Jorge Perelló, 319 Provenza, Barcelona.
Vice-Secretario: Dr. A. Pinart.
Vocal: Dr. J. M. Ferrando.

SOCIEDAD NACIONAL DE CIRUGIA OF CUBA.

Presidente: Dr. Reinaldo de Villiers.
Vicepresidente: Dr. César Cabrera Calderin.
Secretario: Dr. José Xirau.
Tesorero: Dr. Alfredo M. Petit.
Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Fernand Eeman, Ghent, Belgium.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa. U. S. A.
Meeting: Second International Congress of Bronchoesophagology.
Time and Place: June 15 and 16, 1953, Albert Plage, Le Zoute, Belgium.

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